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CHEMICAL STUDIES ON A CASE OF BICHLORIDE POISONING.

By D. Sclater Lewis, M. D., and T. M. Rivers, M. D.

(From the Medical Clinic of The Johns Hopkins Hospital, Baltimore.)

Owing to the frequency of poisoning by mercuric chloride, the clinical picture is well known, and numerous articles are to be found dealing with this aspect of the condition.[1]

The pathological features have also received a considerable amount of attention. The best description of the characteristic sublimate kidney is to be found in Heineke's recent article.[2]

The excellent work of Lieb and Goodwin,[3] and of Lambert and Patterson[4] has done much to place the treatment of the condition on a more substantial foundation. Thanks to their suggestions, we now are able to save a considerable number of cases that almost certainly would have terminated fatally with the old methods of treatment. They have emphasized the value of prolonged and energetic measures directed to the removal of mercury from the body by every means at our disposal. Frequent lavage of the stomach and irrigation of the colon afford the most efficient means of elimination, and should be carried on till mercury can no longer be demonstrated in the washings.[5]

The key-note of the present mode of treatment is *elimination.*

Studies on the metabolism in bichloride poisoning, however, have been of a very superficial character, in most cases confined to a few observations on the level of the non-protein nitrogen of the blood toward the end of longer or shorter periods of anuria. Our chemical studies have been more extensive than any reported in the past, and will, therefore, be given in some detail.

HISTORY.

Med. No. 34753. Mrs. E. D., Age 20; Married. Admitted to The Johns Hopkins Hospital, Sept. 21, 1915. Discharged Oct. 19, 1915.

Complaint.—Bichloride poisoning.

F. H.—Not important.

P. H.—Bichloride douches have been taken for some time, on account of leucorrhœa.

[1] Foster, N. B.: Arch. Int. Med., 1915, XV, 755.
Meyerstein: Münch. med. Wchnschr., 1911, LVIII, 1965.

[2] Heineke: Ziegler's Beiträge, 1909, XLV, 197.

[3] Lieb and Goodwin: Jour. Am. Med. Assn., 1915, LXIV, 2041.

[4] Lambert, S. W., and Patterson, H. S.: Arch. Int. Med., 1915, XVI, 865.

[5] A very sensitive test for mercury in the excreta is described by Vogel (Karl) and Lee (Ivan): Jour. Am. Med. Assn., 1914, LXII, 531.

P. I.—Twenty-four hours before admission the patient took, on an empty stomach, what she thought to be a headache tablet. In half an hour vomiting commenced and persisted till her admission. At first the vomitus was blue; later, colorless. It never contained blood. A physician saw her ten hours after the accident, but did not prescribe. There were several watery stools during the night, but no melena; no abdominal pain. There were severe cramps in the legs. She voided during the morning of the 21st before admission. She walked to the hospital, and on arrival complained of being very nauseated, and of having a metallic taste in her mouth.

P. E.—T. 100°. P. 96. R. 24. (9 a. m.) Well nourished; drowsy; no edema; no evidence of corrosion about the mouth and throat. Tongue heavily coated; breath foul. Lungs clear. Heart not enlarged; soft systolic murmur at apex. Pulse regular. B. P. (Tycos) 105/85. Abdomen scaphoid; moderate epigastric tenderness; liver and spleen not felt. Reflexes normal. Blood: Wassermann negative. Hgb. 95% (Sahli). R. B. C. 5,760,000. W. B. C. 10,880. No urine could be obtained for examination.

Course in Hospital.—Vomiting was persistent during the first 14 days, on three occasions amounting to more than 800 cc. in the 24 hours. Between the third and tenth days, the vomitus contained many blood clots. Diarrhœa was not a troublesome feature, but melena was present for 21 days. Stomatitis was never severe. It appeared on the sixth day, persisted till the twelfth day, and was localized about the openings of the salivary ducts. Anuria began 18 hours after the accident and lasted six days. The patient was catheterized twice during the period, but only a few cubic centimeters of thick slimy fluid were obtained. This did not contain tube casts or any measurable amount of urea, and was not thought to be urine. On the eighth day of the disease, the anuria ceased and the urinary output gradually rose to a high level which was maintained for some time by a forced intake of water. During the anuric period, the patient became more and more drowsy, but this lethargy disappeared soon after the reestablishment of urinary secretion.

The highest temperature was 100.2° F. The pulse was extremely variable on the seventh, eighth and ninth days, but was never above 110. The blood pressure varied from 110/85 to 130/98 (Tycos). Repeated examination of the lungs and abdomen failed to show anything of interest. There was no recognizable edema at any time.

The patient was discharged "well" on the twenty-seventh day. She has been seen a number of times since, and is apparently in perfect health.

TREATMENT.

The treatment may be summarized as follows:

1. Gastric lavage twice or thrice daily for four days.

2. Rectal saline injections—250 cc. physiological salt solution, with 12 gm. sodium bicarbonate every three hours for three days. For the next 12 days, normal salt solution was given by proctoclysis, in amounts averaging 1050 cc. daily. On one of these days, the drip was omitted entirely because of incomplete absorption.

3. On the eighth day, 300 cc. of a 5 per cent solution of glucose were given intravenously, in part for its diuretic effect, in part for the protein-sparing action of the carbohydrate. On the ninth, tenth and eleventh days, respectively, 500 cc. of a 10 per cent solution were given.

4. Water was forced by mouth from the onset. It was vomited at first but was retained in large part after the twelfth day.

5. Sweat baths were given twice daily for 14 days, and were always followed by satisfactory reactions.

6. Diet: Milk was given after the fourth day, a meat-free diet on the seventeenth day, and full diet was allowed after the twenty-sixth day.

LABORATORY FINDINGS.

The chemical studies were of particular interest. The patient was perfectly conscious during her entire illness, and able to coöperate to the fullest extent. The coöperation of the nursing staff was excellent, and we have every reason to believe that the specimens obtained were complete.

The facts presented fall under three main heads:

A. Chemical Studies of the Blood.

B. Chemical Studies of the Urine.

C. Observations on the Acid-Base Equilibrium of the Body.

A. Chemical Studies of the Blood.

The total non-protein nitrogen[6] and urea nitrogen[7] of the blood, and the sodium chloride[8] content of the plasma, were determined at frequent intervals. These values appear in Table I. The blood sugar[9] was estimated on two occasions.

TABLE I.—ANALYSES OF BLOOD.

Date.	Day of disease.	T. N.-P. N. mg. per 100 cc.	Urea nitrogen. mg. per 100 cc.	Percentage of T. N.-P. N. present as urea Nitrogen.	Sodium chloride of plasma. mg. per 100 cc.	Sodium chloride threshold. mg. per 100 cc. Plasma.	Blood sugar. mg. per 100 cc.	Ambard's coefficient of urea excretion.	Phthalein output in 2 hours.
Sept.									
22	3	81	55	67.8	....	....	....	∞	....
24	5	147	100	68.0	455	....	....	∞	....
27	8	184	133	72.3	410	....	....	....	....
28	9	173	142	82.0	402	....	199	....	....
29	10	178	148	83.2	385	362	....	1.09	....
30	11	170	141	83.0	390	379	....	1.36	±
Oct.									
2	13	125	102	81.6	463	455	....	0.55	[14] 11.5%
4	15	78	55	70.5	530	521	....	0.408	[15] 27.5%
6	17	42	23	54.8	587	572	147	0.186	[15] 35.0%
8	19	26	11	42.4	587	568	....	0.124	....
13	24	22	8	36.4	595	569	....	0.06	45.0%
18	29	22	11	50.0	....	....	....	0.11	....

[6] Day of disease on which test was done.

1. *The Total Non-protein Nitrogen and Urea Nitrogen.*—Foster,[10] Phillip,[11] Woods,[12] and Myers and Lough[13] have published isolated observations on the non-protein nitrogen in bichloride poisoning. Their results were usually obtained

[6] For the T. N.-P. N., a modification of Folin's micro-method was employed. Following Dr. B. B. Turner's suggestion, the NH_3 was driven from the digestion mixture by a combination of heat and aeration, the absorption tube being cooled in a water trough.

[7] Marshall's Urease Method: Jour. Biol. Chem., 1913, XV, 487.

[8] Modified Volhard-Charpentier Method. Ambard and Weill: Sem. méd., Mai 8, 1912.

[9] Benedict and Lewis: Jour. Biol. Chem., 1915, XX, 61.

[10] Foster, N.: *Loc. cit.*

[11] Phillip: Med. Klin., 1913, IX, 912.

[12] Woods: Arch. Int. Med., 1915, XVI, 577.

[13] Myers and Lough: Arch. Int. Med., 1915, XVI, 536.

shortly before the exitus of the patient, and are from 20 to 30 per cent higher than ours. Underhill [14] has reported a series of determinations of the blood urea in four cases. In one of these, recovery took place after the urea nitrogen had been over 150 mg. per 100 cc. for 18 days, and on two occasions above 240 mg.

In our case the T. N.-P. N. reached a maximum of 184 mg. per 100 cc. on the eighth day. It remained stationary for four days and then rapidly fell to normal. The curve of the urea nitrogen was of the same type. It reached its maximum of 148 mg. on the 10th day and then returned to normal.

2. *Factors Influencing the Rate of Accumulation of Waste Nitrogen in the Blood.*—Marshall and Davis [15] have shown that urea is distributed uniformly throughout the tissues of the body with the exception of the bone, fat and kidneys. They have calculated that every gram of urea retained by an individual of 70 kg. will cause a rise of 1.33 mg. of urea per 100 cc. of blood. It is permissible to apply these figures to the urea nitrogen of the blood and to assume that every gram of nitrogen retained as urea will give a rise of 1.33 mg. of urea nitrogen per 100 cc. of blood.

Frequent observations of fasting individuals have shown that the usual excretion of nitrogen in the urine, during the first week, is in the neighborhood of 12-13 gm. *per diem*.[16] Approximately, 85-90 per cent of this nitrogen (*ca.* 11 gm.) is excreted as urea. Our patient weighed 68 kg. on admission and she was starved, as practically no food was retained during the first week of her illness. We are, therefore, justified in assuming that, being anuric, she would retain approximately 11 gm. of urea nitrogen per day, as a result of the tissue destruction which occurs in a normal individual during starvation. This would correspond to a daily rise of about 14 mg. of urea nitrogen per 100 cc. of blood during the first week. The actual daily rise of the blood urea nitrogen between the third and fifth days was 22.5 mg., *i. e.*, 7.5 mg. above the amount calculated to result from simple starvation. This excess must have been due, in large part, to an increased tissue destruction caused by the toxic action of the bichloride.

From the fifth to the eighth days, the rise was only 11 mg. per day—a little lower than the estimated value. The small amount of nitrogen excreted in the urine of the eighth day was quite insufficient to account for the diminished rate of accumulation. It is interesting to note that on two of these days the patient received glucose infusions (80 gm. of sugar in all), and this drop in the rate of accumulation may possibly be due to the protein-sparing action of the carbohydrate.

On the two following days, there was a further drop in the rate of accumulation to 9 and 6 mg. of urea nitrogen per 100 cc. The nitrogen output on these days averaged 3.5 gm. These outputs would have decreased the calculated rise in the blood urea nitrogen to 11 mg. per day. On these days, further glucose infusions were given, and the discrepancy may again be referred to the protein-sparing activities of the carbohydrate.

Austin and Leopold [17] have shown experimentally that the administration of glucose has a distinct protein-sparing action in the acute renal insufficiency following bilateral ligation of the ureters. They have also found that animals receiving glucose have survived the operation longer than those that did not receive it.

At no time, in spite of the long period of anuria, did the blood nitrogen reach the high values reported by other observers, and we are justified in assuming that the glucose was responsible, to a certain degree, for the lower rate of nitrogen accumulation. Mercuric chloride causes an acute toxic necrosis of the renal epithelium, which is followed by complete regeneration if the patient survives the initial period of renal insufficiency. Any procedure, therefore, which tends to reduce tissue destruction and keep the blood nitrogen at a lower level will also tend to prolong the life of the individual and so increase the chances of recovery. The intravenous infusion of glucose in these patients who cannot tolerate carbohydrate by mouth is a means to this end.

3. *Sodium Chloride in the Plasma.*—When one considers the frequency of anuria in bichloride poisoning, and the apparent chloride retention so often seen after the urinary flow is reestablished, one is struck with the rarity of outspoken edema in these individuals. V. Monakow [18] has reported one case with definite edema, but this is unusual. The data presented with his case show that there was no relation between the degree of salt retention and the weight of the patient,[19] and that there was no sweeping out of retained salt, even after the complete restoration of renal function. It is evident from the above that we are not dealing with the type of retention usually present in "chloruremic" nephritis.

Working with rabbits, Heineke and Meyerstein [20] found a marked depression of the plasma chlorides in chromium nephrosis. Values of 360 to 400 mg. per 100 cc. were not at all uncommon, whereas the normal range was between 550 and 610 mg. Animals with these low salt concentrations showed little, if any, edema. If large quantities of salt were administered (1.0-1.5 gm. per kg. per day), the concentration of chlorides in the serum rose rapidly to high values—750 to 790 mg.—and the animals then became very edematous.

Woods [21] has also found an extremely low concentration of sodium chloride in the blood from a case of bichloride poisoning on the fifth day of anuria.

The plasma chlorides have been determined at frequent intervals in the present instance. On the fifth day, the amount

[14] Underhill, A. J.: N. Y. Med. Jour., 1915, CII, 662.

[15] Marshall and Davis: Jour. Biol. Chem., 1914, XVIII, 53.

[16] Munk: Arch. f. Path. Anat., 1893, CXXXI, (Suppl.) 25.
Johannson, Landergren, Sonden and Tigerstedt: Skandin. Arch. f. Physiol., 1896, VII, 54.

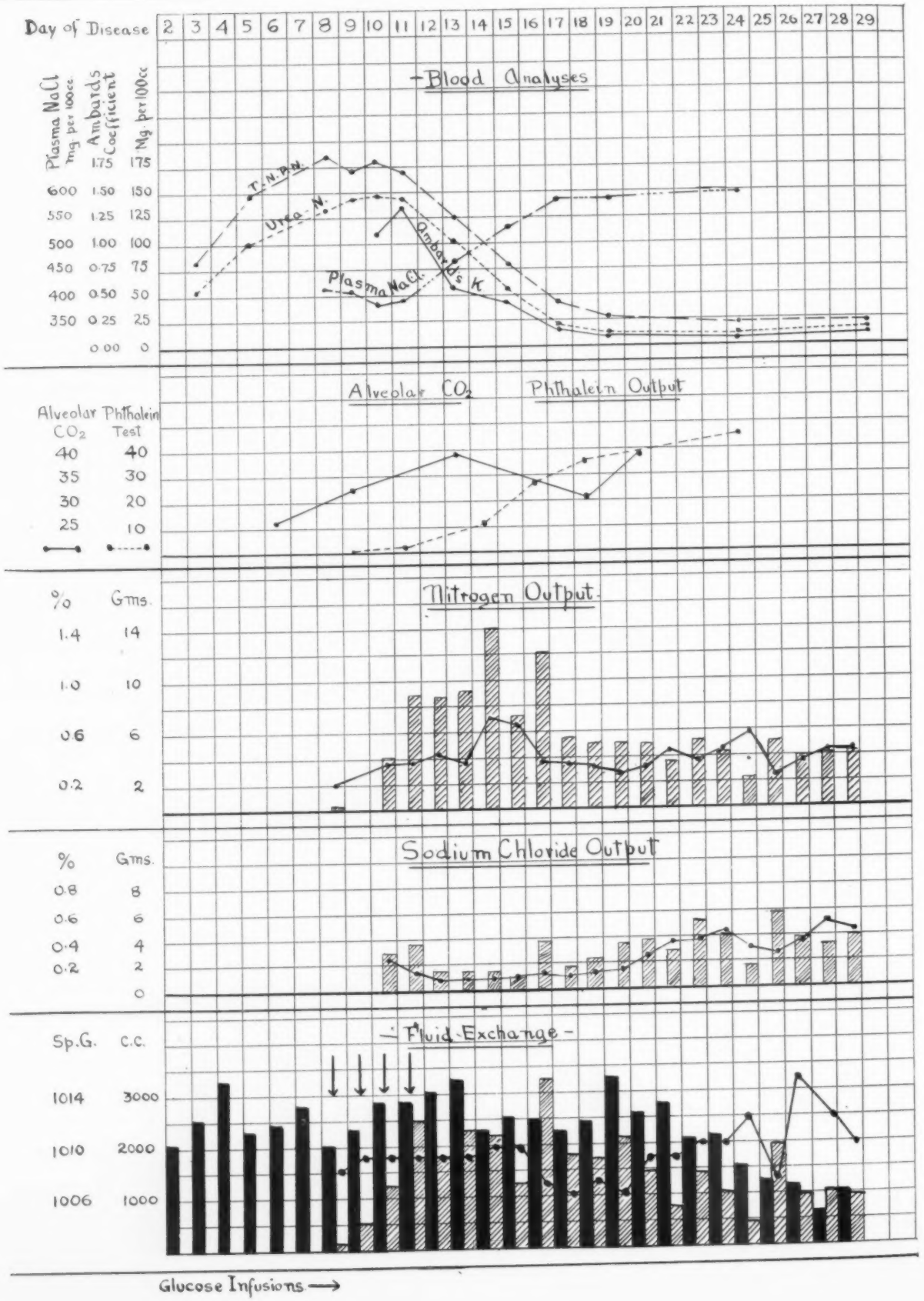
[17] Austin, J. H., and Leopold, S. S.: Proc. Soc. Exp. Biol. & Med., 1915, XIII, 3.

[18] V. Monakow: Deutsch. Arch. f. klin. Med., 1914, CXV, 224.

[19] Javal: Presse méd., Avr. 6, 1914, 477.
Kovesi and Roth Schultz: Pathologie u. Therapie der Niereninsuffizienz, 1904, 157. (Quoted by Ambard, Physiologie norm. et path. des reins, Paris, 1914).

[20] Heineke and Meyerstein: Deutsch. Archiv. f. klin. Med., 1907, XC, 101.

[21] Woods: Arch. Int. Med., 1915, XVI, 577.


Day of Disease
2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29
Blood Analyses
Plasma NaCl mg. per 100cc.
Ambards Coefficient
Mg. per 100cc
600 1.50 150
550 1.25 125
500 1.00 100
450 0.75 75
400 0.50 50
350 0.25 25
0.00 0
T. N. P.N.
Urea N.
Ambard's K
Plasma Na Cl.
Alveolar CO2
Phthalein Output
Alveolar CO2
Phthalein Test
40 40
35 30
30 20
25 10
Nitrogen Output
%
Gms.
1.4 14
1.0 10
0.6 6
0.2 2
Sodium Chloride Output
%
Gms.
0.8 8
0.6 6
0.4 4
0.2 2
0
Fluid Exchange
Sp.G.
C.C.
1014 3000
1010 2000
1006 1000
Glucose Infusions

present was 455 mg. per 100 cc. (normal values, 580-620 mg.). On the eighth and ninth days there was a progressive decrease, and on the tenth day, the plasma chlorides reached their minimum value of 385 mg. per 100 cc. From this time, the quantity gradually increased, but only reached a normal level on the seventeenth day.

Ambard and Weill [22] showed that the urine contained practically no salt if the concentration in the blood were below a certain amount. They called this critical concentration the *threshold of excretion*. Its normal value was found to be in the neighborhood of 562 mg. per 100 cc. of plasma.

The low output of chlorides during the first 2½ weeks of the disease can be explained quite readily by the depression of the sodium chloride of the blood below the "threshold." Reference to the chart and tables will show that the salt outputs were very low until the plasma chlorides had risen above the threshold of excretion. The retention of chlorides in mercuric chloride poisoning is, therefore, of the same general type as that seen in pneumonia.[23] These observations do not explain the mechanism of the removal of sodium chloride from the blood, but they do show that the apparent retention is not due to any impermeability of the kidney to salt.

There are three possible explanations of the low concentrations of the plasma chlorides:

1. The sodium chloride may have been excreted by the alimentary tract and by the skin. A point in favor of this hypothesis is that the amount of sodium chloride decreased regularly during the period of severe vomiting, and the first tendency to rise was shown on the thirteenth day, 24 hours after the vomiting had ceased. The absence of any sweeping out of "retained salt" after recovery, both in this case and in that reported by v. Monakow,[24] would seem to favor the theory of complete removal of salt from the body, rather than any retention in the tissues.

2. The fall of the blood chlorides may have been due to a marked hydremic plethora with a consequent dilution of the constituents of the blood. Georgopulos [25] has suggested this as being the probable explanation of the low concentration of chlorides in the serum of rabbits with a chromium nephrosis. The studies of the rate of rise of the blood urea, and the absence of any very marked change in the blood-count,[26] make this supposition an unlikely one.

3. The tissues may have developed an increased affinity for salt and have withdrawn it from the blood. Such a condition would presuppose the presence of a "dry chloride" retention, as there was no recognizable edema at any time during the patient's stay in the hospital. Had such a dry retention actually been present, there should have been a sweeping-out of salt when normal renal function was reestablished. No such increased elimination of chlorides could be demonstrated.

In the present case, no one explanation can be regarded as being of an entirely satisfactory nature. One can say, however, that there is no salt retention of a type which would contraindicate the use of normal salt solution as a means of forcing fluid on the patient. It is even possible that the body may require salt to make up for the chloride deficit, and that the use of moderate amounts of salt may be a beneficial, rather than a harmful procedure.

4. *The Blood Sugar.*—Frank [27] refers to the frequency of a glycosuria of moderate degree in the experimental nephroses caused by the heavy metals. This glycosuria is caused by a depression of the glucose threshold of the kidney, and, as a rule, no hyperglycemia is associated with it. This lowering of the glucose threshold is interesting in relation to the lowering of the threshold for salt which is also present in this type of renal injury.

We have determined the blood sugar on two occasions, the value of more systematic observations being vitiated by the frequent intravenous infusions of glucose. On each occasion there was a definite hyperglycemia (199 and 147 mg. per 100 cc.), which could be referred to the therapeutic procedures in use at the time.

5. *Ambard's Coefficient of Urea Excretion.*[28]—The return of renal function to normal has also been followed by means of Ambard's coefficient of urea excretion. This coefficient is a constant relation which exists between the concentration of urea in the blood and the rate of excretion of urea in the urine. The normal range of the coefficient is from 0.07 to 0.09. With decreasing renal function, the coefficient shows a tendency to rise, and with increasing function, a tendency to fall.

It is calculated by means of the following formula:

$$K = \frac{Ur}{\sqrt{D \times \frac{70}{P} \times \frac{\sqrt{C}}{\sqrt{25}}}}$$

where K = Coefficient of urea excretion;
Ur = Urea in grams per liter of blood;
D = Output of urea in grams per 24 hours;
C = Concentration of urea in grams per liter of urine;
P = Weight of patient in kg.

The rate of output, D, is corrected for a standard concentration of 25 gm. of urea per liter of urine, and for a standard weight of 70 kg.

In chronic nephritis, coefficients above 0.4 are seen only in the late stages of the disease when there is such outspoken renal insufficiency that death may be expected within a relatively short time. High coefficients may also be obtained in acute nephritis, and in the nephroses, but here they do not have the same significance as in the former cases. In the acute cases, the impairment of renal function may subside and, with improving function, the constant will fall to a more normal figure.

[22] Ambard and Weill: Sem. méd., Mai 8, 1912.
[23] McLean, F. C.: Jour. Exper. Med., 1915, XXII, 366.
Peabody, F. W.: Jour. Exper. Med., 1913, XVII, 71.
[24] *Loc. cit.*
[25] Georgopulos: Ztschr. f. klin. Med., 1906, LX, 411.
[26] First day: R. B. C. 5,760,000. Hgb. (Sahli) 95%. 17th day: R. B. C. 4,176,000. Hgb. (Sahli) 82%.
[27] Frank: Arch. f. exper. Pathol. u. Pharmakol., 1913, LXXII, 387.
[28] Ambard, L.: Compt. rend. Soc. de biol., 1910, pp. 411, 506. Physiol. norm. et pathol. des reins, Paris, 1914.

In the present instance, the coefficient was infinity during the days of anuria. There was no excretion of urea and the fraction

$$\frac{Ur}{\sqrt{D \times \frac{70}{P} \times \frac{\sqrt{C}}{\sqrt{25}}}} \text{ became } \frac{Ur}{\sqrt{0}} = \infty$$

On the tenth day of the disease, the coefficient fell to 1.09. On the eleventh day, in spite of a fall in the blood urea from 148 to 144 mg., there was a slight rise in the coefficient to 1.36. On the following days there was a rapid fall in the constant (0.55, 0.408, 0.186, 0.124, etc.), till a normal level was reached on the twenty-fourth day. During the early stages of recovery there was a close agreement between the variations in the coefficient and in the blood urea. Later, the coefficient's rate of fall became much slower than that of the urea in the blood, and the former did not reach a normal level until a week after the blood urea had been within normal limits.

In the chronic nephropathies, with no marked change in the functional capacity of the kidney from day to day, Ambard [29] and McLean [30] have found that the coefficient is remarkably independent of the concentration of the urea in the blood. With occasional exceptions, the same independence has been noted in a series of observations to be published by one of us in the near future. For every change in the level of the blood urea (Ur), there is a corresponding change in the rate of output (D), with the result that $\frac{Ur}{\sqrt{D}, \text{ etc.}}$ remains constant. Therefore, had the injury to the kidney in this case been a permanent one, the coefficient would have remained at the original level (1.09), despite the changing concentration of urea in the blood. The damage, however, was of a transient nature, and, with the return of the renal epithelium to a normal functional state, the kidney was able to respond more and more fully to the stimulus afforded by the blood urea. As a result, the numerator of the fraction $\frac{Ur}{\sqrt{D}, \text{ etc.}}$ decreased at a greater rate than the denominator, and there was a corresponding drop in the value of K until it eventually reached the normal level.

6. *The Phthalein Test.*—The phthalein test was done according to Rowntree and Geraghty's original technique.[31]

On the ninth day, no recognizable amount of the dye was excreted. Two days later, an unreadable trace was present in the two-hour specimen. Subsequent tests showed a continuous increase in the rate of excretion, and there was a low normal output (45 per cent on the twenty-fourth day) one week after the blood urea and total non-protein nitrogen had shown an apparently normal renal function.

[29] Ambard: Physiologie normale et pathologique des reins, Paris, 1914.

[30] McLean, F. C.: Jour. Exp. Med., 1915, XXII, 212; 366.

[31] Rowntree, L. G., and Geraghty, J. T.: Arch. Int. Med., 1912, IX, 284.

In experimental nephritis it has been shown [32] that the phthalein output drops to its minimum rate of excretion some time before the retention of nitrogen has reached its maximum; that, with a return to normal function, the phthalein reaches its normal rate of excretion before all of the retained nitrogen has been eliminated. The reversal of the order of return to normal in the present instance is probably due, in part, to the low protein intake, and in part to the assimilation of considerable amounts of nitrogen in the repair of tissues broken down during the initial period of starvation and intoxication. As a result, there was relatively little nitrogen to be excreted, and this could be eliminated by the kidney, even though its function was definitely impaired. Retention of waste nitrogen occurs only when the end-products of metabolism accumulate at a greater rate than they can be eliminated by the kidney.

TABLE II.—ANALYSES OF THE URINE.

Date.	Day of disease.	24-hour volume-cc.	Sp. G.	Reaction.	Albumin, gm. per litre.	Total nitrogen.		Sodium chloride.		Glucose.	Microscopic examination.			
						%	Gm.	%	Gm.		Casts.	Epithelial cells.	W. B. C.	R. B. C.
Sept. 21-26	2-7			Anuric.										
27	8	110	1008	Alkaline	3.3	0.23	0.25	....	....	....	++	+++	+	0
28	9	485	1009	"	4.5	.....	.....	.....	.....	.....	++	+++	+	0
29	10	1185	1009	"	3.5	0.335	3.97	0.24	2.84	+	++	+++	+	Few.
30	11	2510	1009	Acid	++	0.356	8.95	0.14	3.51	T.	++	+++	+	Few.
Oct. 1	12	1770	1009	"	++	0.468	8.28	0.08	1.42	0	++	+++	+	0
2	13	2241	1010	"	+	0.338	8.7	0.08	1.79	0	+	+++	+	0
3	14	2095	1010	"	0.75	0.676	14.16	0.08	1.60	T.	+	++	+	0
4	15	1224	1010	"	T.	0.612	7.5	0.08	0.98	0	+	++	+	0
5	16	3260	1007	Alkaline	0.5	0.372	12.13	0.12	3.91	T.	+	++	+	0
6	17	1770	1006	Acid	0.5	0.303	5.36	0.08	1.42	0	0	+	0	0
7	18	1670	1007	"	T.	0.289	4.96	0.14	2.34	T.	0	+	0	0
8	19	2120	1006	"	F.T.	0.239	5	07	0.16	3.39	0	0	+	0	0
9	20	1440	1009	"	T.	0.302	5.21	0.26	3.74	0	0	++	+++	++++	*
10	21	700	1009	"	T.	0.472	3.31	0.38	2.66	0	0	++	+++	++++	*
11	22	1365	1010	"	+	0.383	5.35	0.38	5.39	0	0	++	++	++	*
12	23	962	1010	"	0.25	0.47	4.52	0.42	4.04	0	0	++	+	0	
13	24	430	1012	"	T.	0.58	2.39	0.34	1.46	0	0	+	+	0	
14	25	1940	1007	"	0	0.26	5.06	0.28	5.43	0	0	+	+	0	
15	26	1005	1015	"	0	0.39	3.94	0.38	3.82	0	0	+	+	0	
16	27	1020	1012	"	0	0.43	4.38	0.50	5.10	0	0	0	0	0	
17	28	930	1010	"	0	0.49	4.56	0.42	3.91	0	0	0	0	0	
18	29	*Discharged.*													

* (C. T. M.)

If the protein intake be sufficiently reduced, and if large quantities of carbohydrate be added to the diet, the amount of nitrogen to be excreted may be so reduced that the damaged kidney can excrete it completely and there will be no accumulation in the blood. Under such conditions, the actual state of renal function will not be shown by a simple estimation of the non-protein nitrogen of the blood. It will be more accurately indicated by a comparison of the concentration of this substance in the blood with its rate of excretion in the urine. This can be done most satisfactorily by the determination of Ambard's coefficient of urea excretion, which is absolutely independent of the nitrogen intake of the individual. In reality, the phthalein test is also based on the same fundamental principles. A known amount of the dye is injected and its rate of excretion determined. The test is, therefore, a comparison between the rate of excretion of the dye and the concentration of the dye in the body.

[32] Fitz, K.: Arch. Int. Med., 1915, XV, 524.

The results in the present instance are in strict accord with the above, and we may assume that the slower return to normal of the phthalein excretion and the coefficient are more accurate indications of the establishment of a normal renal function than the more rapid drop of the blood urea and T. N.-P. N.

B. Studies of the Urine.

1. *Fluid Exchange.*—The average fluid intake during the initial period of anuria was 2500 cc. daily. We were not able to make any exact records of the output in sweat, stools or vomitus during this period. From a study of the fluid exchange during the subsequent period of elimination, and from the absence of edema, we are justified in assuming that a considerable portion of the fluid taken during the first week was eliminated by the gastro-intestinal tract and by the skin.

Between the eleventh and twentieth days, there was a consistent polyuria; this was caused more by the high fluid intake than by any marked retention of fluid during the week of anuria. During the third week the urinary output reached more normal values, corresponding with the gradual diminution of the amount of fluid taken by the patient.

2. *Specific Gravity.*—The specific gravity curve is of considerable interest. It showed a marked degree of fixation in the early stages of returning renal function, the maximum range being from 1008 to 1010. This might not have been of any particular importance had the daily output of urine also remained constant, but during this period there were great changes in the daily volumes of urine passed (287 to 2500 cc.), without any corresponding variation in the specific gravity.

Mosenthal [32] has shown that considerable information regarding the condition of renal function may be derived from a study of the specific gravity of the urine. He has found that, in nephritis, marked fixation, at a low level, occurs only in the severest types. This fixation is characteristic of the kidney which has absolutely no reserve power, and which is compelled to function constantly at its maximum capacity in order to eliminate the end products of metabolism.

A reference to the curve of blood urea and total non-protein nitrogen will show that it was only after these values had fallen to normal that any variation occurred in the specific gravity. At first there was a drop in the specific gravity, showing that the amounts of waste material to be excreted had fallen below the level necessary to stimulate the kidney to its maximal concentrating power. During the second and third weeks, the specific gravity showed greater variations from day to day, and this was coincident with the improvement in renal function. Towards the end of the period of observation, the kidneys began to show relatively normal concentrating power, considering the fact that the patient's diet was poor in protein and rich in carbohydrate.

3. *Albumin.*—On the first three days after the cessation of anuria, albumin was present in considerable amounts. As the volume of urine increased, the amounts fell rapidly, and no albumin was found in the urine after the twenty-fourth day.

[32] Mosenthal, H. O.: Arch. Int. Med., 1915, XVI, 733.

4. *Sodium Chloride.*[34]—The output of salt was extremely small during the first week of urinary excretion. We have already discussed the underlying causes of this apparent retention and have found it to be due to the marked lowering of the concentration of the plasma chlorides. With the return of normal concentrations of sodium chloride in the blood, the usual output of chlorides in the urine was reestablished. There was no sweeping out of chlorides after the recovery of normal function.

5. *Nitrogen.*—The total nitrogen excreted in the urine was determined by the Kjeldahl process, after the removal of the albumin by means of heat and acetic acid. There was an initial period of increasing excretion, followed by a few days of maximal renal activity during which the retained nitrogen was eliminated, and, finally, a period of approximate nitrogen equilibrium.

The functional power of any tissue may be gauged by its ability to respond to stimulation. The non-protein nitrogen of the blood may be regarded as the stimulus applied to the kidney, and the quantity of nitrogen excreted as the response of the kidney to that stimulus. It is also evident that the response to a given stimulus will be greater or less, according to the functional capacity of the renal epithelium. A comparison of the non-protein nitrogen of the blood from the eleventh to the seventeenth days with the nitrogen output during the same period is of considerable interest. During these eight days, the non-protein nitrogen fell from 170 mg. to 42 mg. per 100 cc., but the rate of output of nitrogen did not show any downward change at all comparable with the marked fall in the nitrogen of the blood. This is a direct evidence of the improvement of renal functions as, with a constantly decreasing stimulus, the renal epithelium was able to excrete amounts of nitrogen as great or greater than had been possible in the earlier periods with much higher grades of stimulation.

C. Observations on the Acid-Base Equilibrium.

Although the presence of acidosis in uremia was recognized over 25 years ago by von Jaksch,[35] but little has been added to our knowledge of the subject until very recently.

In 1912, Straub and Schlayer [36] determined the CO_2 tension of the alveolar air in a series of cases of uremia, and found a fairly uniform decrease in the carbon-dioxide tension. They concluded that a definite acidosis was present in every case of uremia and that the uremic picture was due in large part to this acidosis. From the results of an exhaustive study of the blood and urine, Sellards [37] decided that an acidosis was present in many of the severer cases both of acute and chronic nephritis, as well as in outspoken instances of uremia. Palmer and Henderson [38] also reached the same conclusions from studies of the acidity of the urine, and the rate of excretion

[34] Sodium chloride was estimated by the Volhard method.

[35] Von Jaksch: Ztschr. f. klin. Med., 1889, XIII, 350.

[36] Straub and Schlayer: Münch. med. Wchschr., 1912, LIX, 569.

[37] Sellards, A. W.: Bull. J. H. H., 1914, XXV, 141.

[38] Palmer, W. W., and Henderson, L. J.: Arch. Int. Med., 1915, XVI, 109.

of ammonia in nephritis. They found a marked decrease, both in the percentage of total nitrogen excreted as ammonia and in the absolute amount of ammonia excreted in the severer cases. The low excretion of ammonia was considered a protective mechanism. The damaged kidney could not excrete the acid radicals, and consequently ammonia was retained by the body to neutralize them. In their opinion, the acidosis of nephritis was due wholly to the retention of acid, and not, as is the case in diabetes mellitus, to the overproduction of acid bodies. Howland and Marriott[39] are of the same opinion. Peabody[40] has repeated Straub and Schlayer's work, and has found that the CO_2 tension of the alveolar air gives evidence of a severe grade of acidosis in many cases of uremia. He has shown that the acidosis has no part in the causation of uremia, but is merely an attendant symptom. He has found it possible to control the acidosis with infusions of sodium bicarbonate, but, while the air hunger may disappear, there seemed to be no tendency for the coma or the uremic symptoms to be changed in any way.

In the present instance, the examinations of the acid-base equilibrium were decidedly interesting, although apparently contradictory in nature. The carbon-dioxide tension of the alveolar air was distinctly low during the period of anuria, being 26.8 mm. of Hg. on the sixth day. Three days later (two days after the cessation of anuria) it had risen 33 mm., and on the thirteenth day had reached a normal value of 39 mm. Five days later, the CO_2 tension dropped to 30.6 mm., but again rose to normal 48 hours later.

The low CO_2 tension on the sixth and ninth days gives evidence of a marked acidosis at that time. This finding is in accord with the ideas of Palmer and Henderson that the acidosis of nephritis is due to an imperfect excretion of the acid radicals, as, at the time of severest acidosis, there was anuria with complete retention of the end-products of metabolism usually excreted in the urine.

Studies of the ammonia excretion on the seventh and ninth days also seem to agree closely with Palmer and Henderson's findings, the amounts excreted being small, both in absolute amount and also in relation to the total nitrogen of the urine.

Sellards, on the other hand, has shown that, in the absence of a cystitis, an alkaline urine is never excreted during acidosis. Yet, coincidentally with the above findings, which would seem to indicate the presence of a outspoken acidosis, the urine of the seventh, eighth and ninth days had a definitely alkaline reaction. On the eleventh and twelfth days, the urine was distinctly acid and the output of ammonia dropped still further, so that only 1.5 per cent and 2 per cent of the total nitrogen was excreted as ammonia. This should have indicated an increasing degree of acidosis, but in spite of this, the carbon-dioxide tension of the alveolar air rose to normal, showing a disappearance of the acidosis, as measured by this means.

Studies of the hydrogen-ion concentration of the blood[41] did not reveal a definite acidosis at any time during the patient's stay in the hospital.

In the interpretation of our findings the main difficulty lies in correlating the depressed alveolar CO_2 tension and the low ammonia coefficient with the excretion of an alkaline urine. Reference to the literature[42] shows that urine excreted during the early stages of mercury poisoning is frequently alkaline, even in the absence of alkaline therapy. The cause of this alkalinity may lie at any one of three different levels; in the tissues, in the bladder and lower urinary tract, or in the kidney. The lowered tension of the alveolar CO_2 can be accepted as proof of an acidosis of the blood and also presumably of the tissues. The low ammonia coefficient absolutely rules out the presence of a cystitis with an ammoniacal fermentation of the urine. It is quite possible that the kidney is the principal factor in the production of these anomalous results. In health the kidney is more permeable to acid than to alkali, and secretes an acid urine from an alkaline blood. In chronic nephritis this permeability to acid is definitely impaired,[43] more and more acid radicals are retained in the body, and eventually an acidosis is set up. Under such conditions, however, an acid urine is the rule, and not one of alkaline reaction. The lesion in the "sublimate" kidney is quite unlike that seen in chronic nephritis. The damage is confined almost entirely to the tubular elements, and there are large deposits of the alkaline earths, in which calcium salts predominate.[44] It is possible that in some stages of the intoxication such a kidney might be relatively impermeable to acids, but still permeable to bases, with the result that an alkaline urine might be excreted, even in the presence of a mild acidosis. This suggestion is advanced merely as an hypothesis to explain the presence of an alkaline urine at a stage of the disease when other findings pointed to a definite condition of acidosis.

With the return of a normal kidney function all signs of acidosis disappeared.

SUMMARY.

1. The necessity of prolonged and vigorous treatment of every case of bichloride poisoning cannot be too strongly emphasized. Many apparently moribund individuals have been saved by properly directed, and vigorously pushed therapeutic measures. Death should be the only indication for a discontinuance of treatment prior to the complete recovery of the patient.

2. Retention of waste nitrogen is undoubtedly a factor in the early fatal issue of these cases. There are rarely any signs of uremia. The protein sparing powers of the carbohydrates

[39] Howland, J., and Marriott, W. McK.: Bull. J. H. H., 1916, XXVII, 63.

[40] Peabody, F. W.: Arch. Int. Med., 1914, XIV, 236; 1915, XVI, 955.

[41] Levy, Rowntree and Marriott: Arch. Int. Med., 1915, XVI, 389.

We are indebted to Dr. J. H. King for the determination of the alveolar CO_2 and to Dr. Levy for the estimation of the H-ion concentration of the blood.

[42] Pfeiffer: Deutsch. Archiv. f. klin. Med., 1907, XC, 591.

[43] Palmer and Henderson: *Loc. cit.*

Howland and Marriott: *Loc. cit.*

[44] Heineke: *Loc. cit.*

Saikowski: Virchows Arch., 1866, XXXVIII, 346.

are of the greatest value in delaying the appearance of the extreme grades of nitrogen retention which usually precede death. If carbohydrate cannot be retained by mouth, glucose may be given intravenously in a 10 per cent to 50 per cent solution. In addition to its protein-sparing action, the glucose itself acts as a mild diuretic.

3. It is probable that alkalis have a decidedly beneficial action. Macnider[5] has recently pointed out that they are capable of protecting the kidney from the full effects of uranium intoxication. It is possible that large doses of sodium bicarbonate given intravenously soon after the taking of the poison would exert a similar protective action in bichloride poisoning. The diuretic action of the alkalis is well known.

4. There is a very close agreement between most of the functional tests. Closer examination, however, shows that tests of retention or of elimination alone do not reflect the actual state of renal function as accurately as do these tests, which compare the rate of excretion of various substances in the urine with the concentration of the same substances in the tissues of the body. The coefficient of urea excretion and the phthalein test can be depended on to give more accurate information regarding certain phases of renal function (the excretion of nitrogen and water) than can be obtained either from the determination of the level of the T. N.-P. N. in the blood or of the amounts of nitrogen excreted in the urine.

5. The specific gravity curve is a valuable index of renal function. Fixation of the specific gravity is seen only at the height of renal insufficiency. The variations in the concentration of the urine become more and more marked as renal function approaches normal.

6. The acute course of the intoxication is shown by the functional tests. Even after six days of anuria, the kidneys regained a practically normal functional capacity within 12 days of the reestablishment of urinary secretion. All trace of anatomical damage disappeared from the urine in less than three weeks.

7. The low value of the chlorides in the plasma explain the apparent retention of salt after the secretion of urine has been resumed. The retention is of the same general type as that seen in pneumonia. It is not due to any impermeability of the kidney to sodium chloride.

8. The lowered tension of the alveolar CO_2 would seem to indicate the presence of an acidosis during the early stages of the intoxication.

[5] Macnider: Jour. Exp. Med., 1916, XXIII, 171.

DISCUSSION.

DR. L. G. ROWNTREE: Just a word concerning this alkaline treatment, which is based upon the work of Macnider, whose results have not yet been published. During the summer he informed me that in uranium-poisoned animals the administration of bicarbonate of soda does away with the anuria which follows in these animals on the subsequent administration of an anæsthetic. He also found that some of his animals under alkaline treatment show very slight or absolutely no pathological changes following the ingestion of an amount of uranium capable of producing very striking pathological and functional changes when sodium bicarbonate is not used. The use of the alkali in this case was probably a very important factor in the recovery of the patient.

The lay press has very recently called attention to this alkaline treatment, which, it says, is being used with success in Cincinnati by Fisher in poisoning with bichloride of mercury.

DR. JANEWAY: In addition to the very great scientific interest of these studies on this patient, they emphasize the very great importance of proper treatment of cases of bichloride poisoning. These cases are so common nowadays that it is important for the medical profession to understand as well how to treat them as the laity now understand how to acquire the condition. It is equally essential that every hospital should be prepared to treat cases of bichloride poisoning properly immediately they are received. It is up to every practitioner either to know how to treat these patients or to get them promptly to the nearest hospital that does.

THE FOLLOW UP SYSTEM.

THE METHODS EMPLOYED UPON THE SECOND SURGICAL DIVISION OF THE NEW YORK HOSPITAL.

By FREDERIC W. BANCROFT, M. D., New York.

The compilation of statistics has been important in the advancement of medical and surgical knowledge. Nevertheless, full advantage has not been taken of the opportunities and possibilities in this respect. Buried in the dust in countless hospitals is the labor of innumerable hospital internes whose case histories contain much valuable information. Moreover, too often, memory alone has been depended upon for recording valuable data. However, in the last ten years the classification of histories and compilation of statistics, based thereon, have come to be valued increasingly. Yet one striking deficiency has prevailed—the late results of operations have not been recorded. In the last analysis the end results are the best guide for the surgeon. Codman of Boston was the pioneer in developing and emphasizing this feature. He has made an active and widespread effort to stimulate hospital staffs to make and record post-operative observations.

The conscientious management of a surgical service demands not only a detailed knowledge of the immediate results of operative treatment, but also the ultimate or end results.

In general, until recently, only spasmodic attempts have been made to ascertain the results after patients have left the hospitals. Most surgeons are convinced that all patients should be followed for at least one year. Many patients, who are considered not of sufficient interest to trace, are often the very ones who present unsatisfactory late results.

Such a "follow up" system should prove of inestimable value to the patient, to the surgical staff, and to the hospital.

A. Advantages to the Patient.

There are two main types of cases referred to a hospital for surgical treatment:

1. Those patients for whom operation is necessary to save life.

2. Those for whom operation is indicated to improve health and economic efficiency. It is to this second class that the data obtained from a follow up system are most important.

For instance, a patient suffering from hernia applies to a hospital for advice. He should be informed as to the percentage of risk that is attached to such an operation; how long he will have to remain in the hospital; how long it will be before he is able to perform his regular work, and how long it will be before he reaches the maximum efficiency. He should also be informed what percentage of cases recur. With these data he may make his decision in regard to the operation.

A woman who suffers from lacerations received at child-birth should know not only the immediate mortality of the operation, but also how long she will be confined to the hospital, how long it will be before she is able to perform her housework, and what effect the operation will have upon future pregnancy and delivery.

The study of the end results will aid the surgeon in the advice he gives the patient. He will recognize that all operations are not successful, and that in certain types of patients a given operation will succeed, while in others it will fail.

B. Advantages to the Surgeon.

The operative policy of a surgical service is directly dependent upon the study and analysis of the late results of operations. If it is found that a certain procedure is not giving permanent relief, it will stimulate the members of the staff to devise means that will improve the results.

In the year that this system has been in operation at the New York Hospital, it has been found that a certain number of patients, operated upon under the diagnosis of chronic appendicitis, have not been relieved of their original complaint. Certain members of the staff are now making a careful analysis of all cases diagnosed as chronic appendicitis, with the object of determining wherein lies the fault. Such a study can be made only by careful analysis of the histories, physical examinations, laboratory and X-ray data, operative findings, and the *end results of a large series of cases.*

C. Advantages to the Hospital.

In the reduction of the number of cases returning for secondary operations and the reduction of the number of surgical complications, such as sepsis, phlebitis, cystitis, etc., lies an excellent means for utilizing hospital funds for the care of the greatest number. A bed may be monopolized for months by a single patient with local sepsis, which otherwise would be occupied by several patients in rotation. Every patient day lost in a charitable hospital, by these complications, should be multiplied by the daily per capita expense. In the weekly discussions described later, all cases of infection, sepsis, etc., are discussed and methods are sought for their prevention in the future.

We have found occasionally that dissatisfied patients have left the hospital and have gone elsewhere for treatment; but upon receipt of our postals and letters they have returned, and in many instances we have been able to correct their erroneous impressions. Every cured and satisfied patient leaving the hospital is an asset, every unimproved or dissatisfied patient is a liability.

Before discussing the details of "The Follow Up System," I wish to mention the weekly conferences held by the second surgical division. These include the report of the return "Follow Up" cases for the previous week. This review is essential for the improvement of the work of the surgical organization.

Once a week grand rounds are conducted by Dr. Pool, the attending surgeon, with the staff and clinical clerks. Every case is dressed. The treatment of the wounds is discussed and a permanent record made on the bedside charts of the condition of each patient and wound. Following the rounds, a conference is held by the entire staff, including attendants and internes. A full discussion is encouraged in regard to—

A. Details of the cases noted on rounds. Many questions bearing upon the progress of the case; the diagnosis or operation may call for discussion either of an explanatory or of a critical nature.

B. A detailed, tabulated report and discussion of the operative results of the preceding week. (*Cf.* below.)

C. Diagnostic errors of the preceding week.

D. A detailed, tabulated report and discussion of the end results in the return cases examined on the previous Sunday.

Let us consider topics B, C and D in detail:

B. Operative Results.

The operative cases of the week are discussed; unfavorable results and unfavorable developments are analyzed, and an effort made to determine the cause of any unsatisfactory condition. The poor results are tabulated as advised by Codman as follows:

1. Errors due to lack of technical knowledge or skill (E-s).
2. Errors due to lack of surgical judgment (E-j).
3. Errors due to lack of diagnostic skill (E-d).
4. The patient's enfeebled condition.
5. The patient's unconquerable disease.
6. The patient's refusal of treatment.
7. The calamities of surgery, or those accidents and complications over which we have no control.

For example: In the permanent records, sudden and unexplained deaths are marked as surgical calamities (S-c). Infections and hæmatomas as errors of surgical skill (E-s).

C. The Diagnostic Record of the Week is then Considered.

Errors of diagnosis (E-d) are referred to the operator for explanation, and a general discussion of the case follows. This is an extremely important feature of the work. It is believed that such an analysis and discussion will lead to greater care in arriving at a correct and exact diagnosis before operation.

will emphasize diagnostic mistakes, and call attention to means of avoiding them in future cases.

To obtain an exact diagnostic record, we employ the following method: The anæsthetist, prior to the beginning of each operation, records the ante-operative diagnosis on the anæsthesia chart. The operator is entitled to record his diagnosis up to the time of making his initial incision; after that it cannot be changed. At the end of the operation a revised diagnosis is recorded.

In the cases in which the diagnosis at the close of the operation is still in doubt, as in tumors, and in some cases with acute abdominal symptoms where no lesion has been found, final decision as to diagnosis is deferred until it is determined by the course of the case, or by the pathologist's report.

D. THE RESULTS IN THE CASES WHICH HAVE BEEN EXAMINED ON THE PREVIOUS SUNDAY MORNING ARE ANALYZED.

Those recorded as "fair" and "poor" are discussed, and, if necessary, the original histories of the cases are reviewed. The cause of failure is sought and a plan for avoiding similar failures in the future is considered. The errors or failures are grouped according to the classification given above in section C.

A follow up system, to be effective, must be so adapted that it automatically follows for at least one year all cases leaving the hospital, and, in certain selected cases, for a much longer period of time.

The present system is so constituted that all patients on leaving the wards are requested to return for observation at the end of three months; if at that examination there is no need for earlier observation, they are requested to report again at the end of nine months. In this manner all cases are followed for at least one year.

The forms used are modelled, with slight variations, after those designed by Corscaden of the Presbyterian Hospital, New York. His work in this direction cannot be too highly commended.

NEW YORK HOSPITAL.

SECOND SURGICAL DIVISION.

FOLLOW-UP SYSTEM.

1. Each morning the charts and bedside cards of the patients to be discharged that day are gathered by the stenographer from the wards. The names and numbers of the patients are entered on card A. These are filed alphabetically.

2. Card B is then filled out for each patient, with his name and number on one side, and the date and hour at which he is to return for examination on the other. These are then attached to the bedside cards and sent back to the wards to be given to the patients on their departure.

In filling out card B, the date upon which a patient is to return for examination is figured out approximately three months from the date of discharge. As each attending has certain Sundays in each month when he is on duty, the patients operated upon or treated by him are requested to return on those Sundays.

3. The name and number and date of examination of each patient is then entered on card C, which is filed according to the date upon which the patient is to return.

4. The data for filling out card D and its carbon copy E is taken from the chart and bedside card of each patient discharged. This is done every Monday morning for the discharges of the previous week.

Card D is then filed numerically.

Card E is attached to card C in the calendar file.

5. At the end of each week, card C and sheet E of all patients due to report on the following Sunday are taken from the calendar file, placed alphabetically in boxes, and sent to the examining room.

A red signal is placed on the corresponding master sheet D in the numerical file to signify that the carbon copy of the case has been sent to the examining room. This signal is removed on return of sheet E and card C.

If the patient has returned for examination, the master sheet is then filed away numerically and the date card filed according to the date upon which he is to return the second time. If the patient did not return for examination, a yellow signal is placed on the master sheet, signifying that a letter is to be written requesting the patient to return the following Sunday. If he returns, the master sheet is filed away numerically. If, however, he does not come to this second appeal, a green signal is applied to the master sheet, signifying that the case has been reported to the Social Service.

6. As each patient comes up for examination on Sunday morning, the examining doctor makes note of his observations on card C, also the approximate time of the patient's next return.

7. The day after the examination the boxes are returned to the filing room. The doctor's notes of his examination on card C are then typewritten on the inside of the master sheet D and its carbon copy E in the space marked F.

A new card C is made out with the patient's next date of examination. The old card C is destroyed.

C and E are then filed again under the next return date on the calendar file.

The master sheet D is returned to the numerical file.

When the folder F is filled, it is torn off and inserted in the original history of the patient, and a new folder placed in the numerical file.

8. Each Monday a postal card G is sent out as a reminder to all patients due to report the following Sunday.

9. A letter H is sent to the patients who did not return for their first examination.

A yellow signal is placed on the master sheet D in the numerical file, signifying that a letter has been sent to the patient. These signals are removed when the patient reports or when the case is given to the Social Service.

The following Sunday the carbon sheet E and card C of all patients to whom letters have been sent are included in the cases sent down to the examining room.

10. If the patients to whom letters have been sent do not report in a week's time, either by letter or in person, the card C

CARD A.

.....

.....

.....

Form 259

NAME HISTORY No.

CARD C

DATE OF EXAMINATION DATE TO RETURN

Name	Age	M. W. S.	Date of Admission	Date of Discharge	Hosp. No.
Addr. of Pat.	MASTER SHEET D		1		
Name and Addr. of Pt's Phys.			2		
Name and Perm. Addr. of Fr'nd			3		
Preop. Diag.		2		3	
Post-op. Diag.					
Came for relief of					
Operator	Anes.				
Operation					
Condition of wound					
Compl. of Convales.					
Path. No.					

POSTAL CARD G.

NEW YORK HOSPITAL

8 WEST 16th STREET

Will you return to the Hospital on Sunday..... .
at.....A. M., as we desire to note the progress of our patients after they leave the hospital. Advice will be given you in regard to your condition.

Bring this card with you. If you cannot come, please write.

.....
Attending.

Dear Doctor: CARD I.

The following cases operated upon or treated by you are due to report on:

NAME	DIAGNOSIS	TREATMENT	HISTORY No.

Surgical Record Clerk.

and the sheet E are sent to the Social Service nurse, the case to be investigated by her and the patient induced to report for examination if possible.

When the case is given to the Social Service nurse a green signal is placed on the master sheet, which is removed on the receipt of a report from the Social Service nurse or return of the patient.

11. The attending surgeon receives a list (Card I) of the patients operated upon or treated by him, who are due to report for observation.

WEEKLY REVIEW OF CASES.

Sunday morning was chosen, because it is the day on which most people of the working class are free. The patients are requested to report at 9.30 a. m. One of the attending surgical staff, three internes, and the Social Service nurse, employed for the follow up system, are in attendance. The two junior members of the interne staff take histories of the cases. In order to include the important facts of the history, an outline is furnished with the history cards.

POST-OPERATIVE HISTORY.

1. Local Condition: Pain. Function.

2. Economic: Ability to do full amount of work; and how soon after discharge.

3. General Condition: Comparison of before and after operation. Did the operation or treatment cure the trouble? Weight—loss or gain?

4. Body Functions: Appetite. Digestion. Bowels. Urination. Menstruation. Marital.

The attending surgeon on duty and the house surgeon then examine the patients and make notes upon card C of the Follow Up System, as described above. Immediately following the physical examination, a note is written on the card tabulating the case as "good," "fair" or "poor." It is from this tabulation that the analysis is made for the discussion at the weekly conference.

HELP EMPLOYED FOR SUCH A SYSTEM.

During the year 1915 there were 1344 patients admitted by the Second Surgical Division. One stenographer is employed by the staff; she carries out all of the clerical work of the Follow Up System; besides this, she typewrites the operator's descriptions of all operations.

SOCIAL SERVICE.

When no response has been received from either the postals or letters, the case is referred to the Social Service Department, which has assigned the nurse to trace these cases. Her success depends, to a large extent, upon her energy and ingenuity. In New York, people living in tenements and boarding houses move frequently, and it is often difficult to find them. It may be necessary to determine their nationality and their religion—the nearest church of that denomination will often yield the information desired; or it may be sufficient to inquire from neighboring stores or saloons. If the nurse is persistent, her percentage of returns will be relatively high. If she is unable to persuade patients to return for examination, it is her duty to record whatever information she can acquire.

Now in regard to the practical *modus operandi* of the system, we have found that the personal element is important. If the house surgeon interviews the patients at the time of discharge, and impresses upon them the fact that they may be benefited by the examination, a large number will report. If they return once, they will usually return in response to a second request. In fact, they will often come for examination when not expected, because of some ailment. The following is a report from February 1, 1915, to December 1, 1915:

REPORT OF THE FOLLOW-UP SYSTEM FROM FEBRUARY 1 TO DECEMBER 1, 1915.

Cases to return	808	
Cases returned on first request		399 or 49%
Cases returned from letters		87
Patients reported by letter		87
No. of cases returned and heard from		573 or 70%

SOCIAL SERVICE.

Cases given to Social Service	228	
Return of patient		64 or 28%
Report from nurse		70 or 30%
Not found by nurse	90	
No. of cases returned and reported upon		134 or 58%
Total No. of cases returned		550 or 68%
Total No. of returned, heard from and reported upon by Social Service		707 or 87%

In order to demonstrate the efficiency of the system, we have reviewed the cases of simple inguinal hernia operated upon between February 1, 1915, and September 1, 1915. A complete review would be out of place here.

Let us, however, consider, for example, the economic factor: There were 120 patients with hernia operated upon; of these, 108, or 90 per cent, were traced. Mention was made of the patient's ability to work in 96 cases, or 89 per cent. No note was made in 12 cases, or 11 per cent.

Of these 96 patients:

Eighty-four, or 87.5 per cent, were performing their usual work at the end of three months.

Nine, or 9.4 per cent, were performing light work.

Three, or 3.1 per cent, were unable to work.

Twenty-two patients gave definite data of how soon they went to work after leaving the hospital:

2	went	to	work	in	1	week	after	discharge.
9	"	"	"	"	2	weeks	"	"
1	"	"	"	"	3	"	"	"
1	"	"	"	"	4	"	"	"
3	"	"	"	"	6	"	"	"
2	"	"	"	"	8	"	"	"
2	"	"	"	"	10	"	"	"
2	"	"	"	"	32	"	"	"

In reviewing our cases diagnosed as chronic appendicitis, we found that a considerable number were not relieved in regard to their original complaint.

In order to study these cases, we have adopted a more elaborate history form and complex physical examination. As time goes on it is very probable that certain forms of histories and physical examinations will be developed for different types of cases, so that in studying the late results of a series of cases there will be adequate data for extensive research. Our main error in the first months of the system was a tendency to write insufficient histories and inadequate physical examinations.

In closing, I wish to thank the staff of the Second Surgical Division for the privilege of presenting this report.

The organization of the system was largely due to the efforts of Dr. Pool and Dr. Turnure.

DISCUSSION.

DR. WINFORD SMITH: Dr. Bancroft referred to Dr. Codman of Boston. I had the pleasure of hearing Dr. Codman speak recently on the subject of the End Result System, and whenever you have the opportunity of reading what Dr. Codman has to say about "end results" I strongly recommend that you do so. It is interesting reading and presents some facts that bear thinking over carefully.

We have passed through the sanitary stage in hospitals—the stage when we were all more or less sanitary mad. I have about come to the point of view that we are now going through that stage with regard to the use of the term "efficiency." There is one danger, it seems to me, in the general adoption of follow-up systems. Without in any way meaning to reflect upon the system as outlined by Dr. Bancroft, which has, I think, great merit and which I have observed working most successfully, I do think we ought to bear in mind that it is a very easy matter to be carried away by the advocates of efficiency methods and to install follow-up systems for this or that subject. If a follow-up system collects statistics and data which enable us to know where mistakes have been made, or to devise a better technique, or to finish a job which we had thought was finished, then it is valuable. But a great many of these systems, as I have known them, have been established merely with the idea of doing something in the line of efficiency, and have collected a great mass of statistics, some useful and much of them useless, with the latter obscuring the former. That sort of follow-up system is not of much use, and before we or any other hospital adopt one like it, we should work it out very carefully in its application to our own individual purposes, just as it has been worked out to suit the needs of the New York Hospital. I am sure the society owes Dr. Bancroft a vote of thanks for presenting such an interesting and instructive paper.

WILLIAM BUDD, PIONEER EPIDEMIOLOGIST.*

By ASSISTANT SURGEON-GENERAL W. C. RUCKER, M. S., M. D., GR. P. H.,

United States Public Health Service, Washington, D. C.

So many notable additions to the knowledge of medical science have been made in recent years by laboratory workers that many of us have fallen into the habit of thinking that all medical discoveries must have the microscope and the test-tube as their point of origin. This is in part true, in so far as it relates to the causal agents of disease and their effects upon the body of man and the lower animals, but just as the practice of medicine depends upon knowledge gained at the bedside, so the science of epidemiology rests upon observations made during the spread of disease. When the practice of medicine forsakes clinical study for laboratory investigation the human element is lost. When we limit epidemiological study to the four walls of a laboratory, the scope of its vision becomes narrowed, and facts which could have been gained by personal contact with disease pass unobserved. Not every one has a laboratory at his command, and relatively few have the training which is requisite for good laboratory work, but every physician, no matter what his position nor where he may be located, may conduct valuable epidemiological investigation, if he will but observe what is going on around him. Jenner, the country doctor, gave to the world a sure prophylactic against smallpox. Another country practitioner, working without a laboratory and before the birth of bacteriology as a science, gave to us so clear an outline of the epidemiology of typhoid fever that it still stands as a model and with a few minor additions would summarize our knowledge of the disease.

* An address delivered before The Johns Hopkins Historical Club, Baltimore, Maryland, on March 13, 1916.

William Budd, practitioner of medicine, careful student and logical thinker in the science of epidemiology, was born in September, 1811, in North Tawton, a town on the borders of the beautiful Dartmoor in Devonshire, England. He was the fifth son of Samuel Budd, being one of nine brothers, seven of whom entered the medical profession and became leading practitioners. The family was one of great talent. Five sons graduated at Cambridge. Four of these were fellows of their respective colleges, and all were wranglers. Four of Dr. William Budd's nephews likewise became fellows of colleges in the same university. Both Dr. William Budd and his older brother, Dr. George Budd, the author of the great work on "Diseases of the Liver," were fellows of the Royal Society.

The Budds were descended from Jean Budd, a baron during the time of Charles the Great. As a reward for his military service, Jean Budd was given a domain on the Norman seacoast. His descendant, William Budd, founded the town of Rye, and during the Norman invasion of France he housed the King. His descendant, Richard Budd, had four sons, three of whom became sailors and subsequently settled in the town of Rye in Sussex County, England. John Budd, who succeeded to the barony, came over at the time of William the Conqueror and landed at Rye, where his relatives were then living. He distinguished himself during the Norman invasion of 1066, and married a sister of William the Conqueror. He subsequently became the Earl of Sussex. The family intermarried with the Nevils, the Brownes, and the Montagues, and it was from this distinguished stock that William Budd, the subject of the present sketch, was descended.

With his eight brothers, William Budd was educated in the home of his father, himself a surgeon of no mean ability. In 1827, when he was 16 years of age, he went to Paris. There he studied for four years under Louis Andral, Cruvelhier and Broussais in the Collège de France and the École de Médecine, and, under the tuition of Broussais, first became acquainted with the intestinal lesions of typhoid fever. While in Paris he contracted typhoid, and the leeching and starvation, which was the accepted treatment of the disease at that time, was carried to such a point that he was unable to pursue his studies for several years. In spite of this, he valued bleeding very highly in the early part of his career. Returning from Paris, he continued his medical studies in London and Edinburgh, and subsequently became a pupil of Sir Thomas Watson at the Middlesex Hospital. This is the "Sir T. Watson, Bart.," to whom Budd dedicated his *opus magnum*, "Typhoid Fever, Its Nature, Mode of Spreading and Prevention," "in grateful remembrance of many encouraging words."

In 1838 Budd took the M. D. degree from the University of Edinburgh. His graduation thesis was on "Rheumatism," and secured for him a gold medal. The essay clearly foreshadows the views which he expressed three years later in his paper before the Royal Medical and Chirurgical Society on "Diseases Which Affect Corresponding Parts of the Body in a Symmetrical Manner." This justly famous paper (Medico-Chirurg. Trans. 1842, XXV, 100-166) produced a profound impression. Watson (Principles and Practice of Physic, 3rd edition, p. 694) says that it throws "a strong light upon this perplexed subject, and brings the phenomena, not only of gout, but also of many other important complaints, within the operation of one general, comprehensive and intelligible law." It is interesting to note that on the same evening that Budd's paper was presented, a highly philosophic essay on "The Relation between Symmetry and Disease" was read by no less person than the illustrious Sir James Paget.

After graduation Budd served for a short time as physician to the seamen's hospital ship *Dreadnaught* at Greenwich, but was compelled to resign his post because of an attack of typhus fever of which he almost died. In 1839, during his convalescence, he resided with his brother, Dr. Richard Budd of Barnstaple, and prepared an essay on "Fever," in competition for the Warneford prize of £50. He failed to receive the award, however, because the judges, of whom Sir John Forbes was umpire, were of the opinion that the ideas enunciated in his essay were "too novel and startling."

This is not at all surprising, the medical world at that period being divided into camps, each busily engaged in pursuing its pet theory of disease spread. Brigham, in his "Treatise on Cholera," published at Hartford, Connecticut, in 1832, reflects accurately the beliefs of the times. Speaking of the contagiousness of cholera, he says:

> Upon this delicate and difficult subject medical men are much divided. They may be divided into three classes who hold to different opinions:
>
> 1. The first, though least numerous class among the intelligent medical men, are the exclusive contagionists, who believe that the cholera is communicated from an unhealthy body to a sound one, by approximation, or actual touch. To this class belong also the great mass of the non-medical community—the civil authorities of towns and those who have the power to make and enforce quarantine regulations; regulations which, as regards the prevention of cholera, have been of no use, but, on the contrary, as facts fully exhibit, have been productive of immense evils.
>
> 2. A larger class are the anti-contagionists, who believe that the cholera depends either on a general cause everywhere existing, or arises from numerous causes, the most powerful of which are, exposure to cold and humidity at night, and burning heat during the day, the abuse of stimulating li uors, bad food, want of cleanliness, etc.
>
> Some suppose it is produced by the magnetic influence of the earth, the telluric power. Others think the disease is primitively nervous, and depends on an electro-magnetic cause.
>
> 3. A third, and probably an increasing class, are those who keep to a middle course, between the contagionists and the anti-contagionists, and believe in what is called the doctrine of contingent contagion. These say that, although the disease arises from some aerial or terrestrial influence of which we at present know nothing, and over which we have no control, yet in the filthy hovels of the indigent, in the impure air of crowded apartments, the disease may, and sometimes does, acquire a character of communicability, which it did not at first possess, and of which it is deprived when these circumstances are not present, or when it occurs in well ventilated and cleanly situations.

In the hands of the great Murchison, the anti-contagionist theory that filth was the generator, not the vehicle, of disease was moulded into a dogma to which was applied the name of the Pythogenic theory. In a paper presented to the Royal Medical and Chirurgical Society in 1858, Murchison said:

> In the course of this essay I shall bring forward what I consider positive proofs that this fever (typhoid) is produced by emanations from decaying organic matter, and I would, therefore, suggest for it the appellation of "pythogenic fever."

Charles MacLean, in his work, "Evils of Quarantine Laws and Non-Existence of Pestilential Contagion" (London, 1825), voices perhaps the views of the leading men of the profession at that time:

> The existence of contagion in epidemic diseases is not a question which is now to be proposed for solution. It is already solved. To regard it as problematical or uncertain and disputable, as the pestilential contagionists still do, or affect to do, is to be ignorant of much of what has been done and written on the subject. The impossibility of the existence of contagion universally, in epidemic diseases, is a principle which I have, in various works and upon various occasions, already established upon an immovable foundation.

In the face of such opinions, it is not remarkable that the observations of Budd, then in his 28th year, should receive but scant consideration and that he should labor for more than 40 years on the problem and die long before his beliefs acquired any very general acceptance.

In 1839 Budd returned to North Tawton and entered into practice with his father, and here began those intensive studies which were to be the basis of his work on "Typhoid Fever, Its Nature, Mode of Spreading and Prevention," published over 30 years later. In the introduction to this volume he paints a word picture of the environment in which he then found himself, an environment which is almost duplicated by that of many of the country practitioners of America:

In the early part of my professional life, while engaged in country practice in Devonshire, outbreaks of typhoid fever continually fell under my eye, amid conditions singularly favorable to the study of its origin and mode of dissemination.

Of these outbreaks, the most memorable occurred in the village of North Tawton, where I then lived.

In addition to the advantages enjoyed by country practitioners generally, in the observation of such events, there were others peculiar to the position I then occupied.

Having been born and brought up in the village, I was personally acquainted with every inhabitant of it; and being, as a medical practitioner, in almost exclusive possession of the field, nearly every one who fell ill, not only in the village itself, but over a large area around it, came immediately under my care.

For tracing the part of personal intercourse in the propagation of disease, better outlook could not possibly be had.

At the date of the outbreak in question, the people of the place numbered some eleven or twelve hundred souls.

Of these, a small minority, consisting chiefly of women and children, worked in a serge factory. The rest were employed in agricultural pursuits.

The spot on which the community dwelt is richly endowed with all the natural conditions of health. Built on a dry soil, in the midst of an open and well-drained country, and occupying the side of a hill sloping gently to the northwest, this village had long been justly noted in that part of Devon for the rare healthiness of its site.

What is more to the present purpose is, that it had for many years enjoyed an almost entire immunity from the fever to which it was so soon to pay so large a tribute.

This is the more to be remarked, because there were in the economy of the place, and in the habits of the people, many things which, according to modern views, are hard to reconcile with such a fact. In the first place, there was no general system of sewers. A few houses, occupied by the more opulent, were provided with covered drains, but all these might be counted on the fingers. In the cottages of the men who earned their bread with their hands, and who formed the great bulk of the inhabitants, there was nothing to separate from the open air the offensive matters which collect around human habitations. Each cottage, or group of three or four cottages, had its common privy, to which a simple excavation in the ground served as cesspool. Besides this, it was a part of the economy of all who worked in the fields, as indeed of many more, to keep a pig, one of whose functions was to furnish manure for the little plot of potatoes which fed man and pig alike. Thus, often hard by the cottage door there was not only an open privy, but a dungheap also.

Nevertheless, these conditions existed for many years without leading to any of the results which it is the fashion to ascribe to them.

Much there was, as I can myself testify, offensive to the nose, but fever there was none. It could not be said that the atmospheric conditions necessary to fever were wanting, because while this village remained exempt, many neighboring villages suffered severely from the pest. It could not be said that there were no subjects, for these, as the sequel proved, but too much abounded.

Meanwhile privies, pigstyes and dungheaps continued, year after year, to exhale ill odors, without any specific effect on the public health.

Many generations of swine innocently yielded up their lives, but no fever of this or any other sort could be laid to their charge. I ascertained by an inquiry conducted with the most scrupulous care that for 15 years there had been no severe outbreak of the disorder, and that for nearly 10 years there had been but a single case.

For the development of this fever a more specific element was needed than either the swine, the dungheaps, or the privies were, in the common course of things, able to furnish.

In the course of time, as was indeed pretty sure to happen, this element was added, and it was then found that the conditions which had been without power to *generate* fever, had but too great power in promoting its spread when once the germ of fever had been introduced.

On July 11, 1839, a first case of typhoid fever occurred in a poor and crowded dwelling. Before the beginning of November, in the same year, more than 80 of the inhabitants had suffered from it under my care.

In this environment Budd was able to study the disease in all of its manifestations and to correlate the going and the coming of those who were affected with it. He realized fully the opportunities which the situation offered, and wrote many years later:

Where the question at issue is that of the propagation of disease by human intercourse, rural districts, where the population is thin, and the lines of intercourse are few and always easily traced, offer opportunities for its settlement which are not to be met with in the crowded haunts of large towns.

As a result of his observations at this time he came to the conclusion that the fever was essentially a contagious or self-propagated disease. Yet despite his high enthusiasm in this belief, Budd fully realized that many convincing proofs must be brought forward before his views would be very generally accepted. In 1866 he prefaced his pamphlet on cholera by saying:

I speak still in the interrogative mood, because, however strong the evidence may seem, one is almost afraid to believe in so great a triumph as this, until it is assured by more extended experience. In exact proportion to the importance and magnitude of any given conclusion, the true philosopher, strong though his own conviction may be, is exacting of proof.

In order to appreciate fully the radical nature of such a conclusion, it is necessary to understand the doctrine which was generally accepted at that time. In the Manual on the Practice of Medicine, by Dr. Tanner, which at the time of the publication of Budd's work in 1873 had already reached its sixth edition, the author, in speaking of the doctrine of typhoid fever as a contagious disease, chiefly disseminated through the excreta of typhoid patients, calls it "an elusive hypothesis." The Physician's Vade Mecum, at that time a standard work, dismisses the doctrine with the statement that "observed facts and the few experiments which have been made tend to disprove these views." Elsewhere in the work, the authors, Guy and Harley, state:

Much doubt prevails whether enteric fever be infectious or not. The question really turns upon the existence of a distinct specific organism. Positive proof that it may be conveyed from one person to another is wanting, and certainly the majority of people affected with the disease derive it from the clearest evidence from one and the same source. Those in attendance upon persons suffering from enteric fever sometimes fall ill of the disease, but the source of the disease may be present in the house.

Not only did Budd decide that the disease was contagious, but he came to the conclusion that it was of a specific nature. And in 1873 he clearly prophesied the existence of the bacillus which was not discovered until some seven or eight years later. He said:

It is humiliating to think that issues such as these should be contingent on the powers of a creature so low in the scale of being that the mildew which springs up on decaying wood must be considered high in comparison.

He also came to the conclusion, from the foregoing, that typhoid fever not only propagates itself, but that it propagates no other kind of fever. This was widely at variance from the views at that time held by those who believed in the pythogenic theory of the disease, and as evidence on this point he clearly recognized the period of incubation of the disease, the immunity conferred upon those who had had the disease, and the fact that not all persons living in the same environment contracted the disease.

Very early in his studies Budd recognized that the intestinal discharges played the major rôle in the dissemination of typhoid, and he deduced therefrom:

1st. That as a rule this fever will spread the more, the less perfect the provisions for preventing the discharges from the human intestine from contaminating the soil and air of the inhabited area.

2d. That where these provisions fulfil this end, the disease will show little or no contagious power.

3d. That its tendency to run through families will oftenest take effect where there is only a common privy, least often where there is a well-appointed water-closet. That this tendency will be observed very commonly, therefore, in country places, and comparatively rarely amongst the wealthy inhabitants of large towns.

4th. That, generally speaking, the distribution of the disease will be different in country and in town; that in the country, where there are few or no sewers, and where, consequently, the intestinal discharges accumulate around the infected dwelling, the disease will occur in a thickly clustered manner; that in the town, where these discharges are conveyed, often for long distances, by sewers, the ramifications of which extend through large communities, it will appear in a more scattered form.

5th. That, as what the sewer receives from the fever patient is incomparably more virulent than anything else thrown off by him, the infection (until the true interpretation of the events be known) will appear, for the most part, *as if it had its source in the sewer, and not in the already infected man.*

6th. That in the country, the contagious nature of the fever will be obvious and unmistakable; but that in the town, it will most commonly be masked and obscure.

7th. That in the former, the fever will be epidemic and thickly clustered; in the latter, as a rule, endemic and scattered.

8th. That separation of the healthy from the infected will be of no avail to prevent the spread of the fever, unless it include separation from the intestinal discharges also.

9th. That, for this reason, the severest outbreaks will be seen in schools, barracks, and other large establishments, where a single common privy is often, alike, the receptacle of the discharges from the sick, and the daily resort of large numbers of healthy persons.

In the course of his studies—studies which he never relinquished until forced by the hand of age—he early came to the very logical conclusion that the pollution of water supplies was the great factor in typhoid transmission. In fact, Budd recognized all the media of transmission now known, with the possible exception of flies. In his work on typhoid he says:

One mode of communication has attracted little attention, which it is important, nevertheless, not to overlook; I speak of the tainted hands of those who wait on the sick. Among the poor, and in ways that will suggest themselves, and need not be more particularly described, there is reason to believe that this mode often has a large share in spreading the disease through the family circle. Passing from the hand to other things under contingencies that are not only very conceivable, but are sure now and then to occur, the contagion thus arising may sometimes have a much wider scope. I possess evidence which renders it in the highest degree probable that milk and butter, especially, may become infected in this way.

He clearly recognized the occurrence of milk outbreaks, and in one of his writings thus lays emphasis on the danger of raw milk:

All risk may be abolished by *boiling* the milk, however deeply it may be polluted. Drinking unboiled milk is like eating raw meat, and is open to consequences of the same pathological order. I have no doubt that, if all milk were boiled before being used, a marked diminution in the prevalence of more than one very serious type of the disease would soon follow.

As preventive measures, Budd at all times laid strong emphasis on the disinfection of the discharges of the sick, the isolation of all typhoid patients, the careful and frequent washing of the hands of the attendant, and the boiling of the drinking water. Truly, as he says, "These are golden rules; where they are neglected the fever may become a deadly scourge; where they are strictly carried out, it seldom spreads beyond the person first attacked."

Budd is described by his contemporaries as a strong, hardy looking man of robust body, good height and brave presence. This powerful body, which in his younger days bespoke in every movement the invigorating air of his native Dartmoor hills, housed a mind which was sensitive and emotional almost to a fault. An intimate said of him that he "was possessed of a heart tender as a woman's and gentle as a child's." His energy and industry were unbounded, and the catholicity of his interests was remarkable, almost overwhelming in his later years. That he should be a skilled physician was to be expected. He had been reared in a medical atmosphere and carefully trained both in Great Britain and on the Continent. When he entered practice he was 27 years of age, and when he was 30 his well-matured mind had already arrived at conclusions which at first startled and later modified the views of the medical profession. His practical knowledge of disease, particularly of the communicable diseases, was very great, and included the contagious diseases of cattle, sheep and pigs, as well as those of human beings. His observations on the diseases of animals were very valuable, and in 1863 he published a pamphlet on "Variola Ovina, Sheep's Smallpox; or the Laws of Contagious Epidemics, Illustrated by an Experimental Type." He also studied a disease of swine which he called "pig-typhoid." Rinderpest, or cattle plague, also claimed his attention, and, when the disease first appeared in England,

Budd held emphatically that the only way to stamp it out was to slaughter the animals as soon as they showed symptoms of the disease. His recommendation of "a poleaxe and a pit of quicklime" at first met with great ridicule, particularly at the hands of the *London Times*, but his views were ultimately and very successfully adopted.

His accurate knowledge of French, German and Italian kept him abreast of the medical advances of the Continental schools, while his facile pen led him far into the field of medical literature. He was a skilled artist and a good photographer, accomplishments which were of great assistance to him, particularly in the production of his work on typhoid fever.

In 1841 he removed from North Tawton to Bristol, and speedily assumed a high position among his colleagues. He became physician to St. Peter's Hospital in 1842; physician to the Bristol Royal Infirmary in 1847, and for many years held the chair of medicine in the Bristol Medical School. At the Infirmary he found the pathological museum, which had been started by Robert Smith. With characteristic energy he began the collection of specimens and aided materially in the up-building of the museum. Largely through the championship of Tyndall, who proudly proclaimed himself one of Budd's disciples, he was elected a fellow of the Royal Society—"a rare honor for a provincial medical man" (1870). Tyndall wrote of him:

Dr. William Budd I hold to have been a man of the highest genius. There was no physician in England who, during his lifetime, showed anything like his penetration in the interpretation of zymotic disease. For a great number of years he conducted an uphill fight against the whole of his medical colleagues, the only sympathy which he could count upon during this depressing time being that of the venerable Sir Thomas Watson.

Budd's typhoid studies naturally led him to entertain similar views regarding the transmission of cholera. He held that the contagious agent was present in the dejecta and that by a proper disinfection of all the stools in all the cases, an outbreak of cholera could be suppressed. He applied this method to the eradication of the Bristol cholera epidemic in 1866, with such success that although the disease was introduced at 26 different points in the city, and in severe form, only 29 cases occurred, whereas in 1849 there were 1979 cases.

Dr. Budd says of this epidemic ("Cholera and Disinfection, or Asiatic Cholera in Bristol in 1866," 1871):

Whatever the light by which we view the events, we are thus unavoidably led to the belief that this city (Bristol) owes its exemption from an epidemic of cholera, in the summer of this year (1866), to the specific measures taken to prevent the spread of the malady. Had the principle on which these measures are founded been purely empirical, the facts would have left us no reasonable alternative. But, as we know, the case is far otherwise. By an induction, which for commanding clearness and logical severity is rare indeed in physic, we have arrived at the conclusion that Asiatic cholera is propagated extensively, as I believe, by the rice-water discharges. To destroy these discharges is, therefore, to destroy the seed by which the disease is sown, and, by the very act, to prevent the possibility of a future crop. In Bristol the seed has been destroyed and the crop has failed. What was expected has exactly come to pass. The accordance between theory and fact is not general merely, but very nearly perfect.

The relationship between Budd's work and the cholera investigations of Snow is very interesting. It has been held that to Dr. John Snow belongs the credit for the anti-cholera work, and that Budd added nothing material to the subject. Budd himself was most emphatic in his praise of "Dr. Snow's admirable, long prior, and entirely original researches." The facts, however, are these: Snow proved that cholera was frequently disseminated by drinking water, a discovery the importance of which cannot be overestimated. Budd, however, proved that by the disinfection of the discharges and the isolation of the sick, the pollution of the water could be prevented and the disease checked. So strongly did he believe in the necessity for a full supply of pure water, that he was one of the most zealous promoters of the Bristol waterworks.

Budd was never content to follow the beaten paths, and in his scientific work he exemplified Ptolemy's *dictum* that "He that would follow philosophy must be a freeman in mind." His active brain led him into speculations regarding most of the epidemic diseases, and while the pressure at which he lived left little opportunity for extensive writings, he prepared several short notes which were published, and several longer manuscripts that were discovered after his death, but were never printed. On one occasion he gave a friend, a medical officer about to go to Jamaica, very careful instructions for preventing the spread of yellow fever, and subsequently published these with considerable additional matter in the *Lancet* (April 6, 1861, I, 337). He was firmly convinced that the disease was spread by the excreta and he, therefore, focused his eradicative measures on the disinfection of the discharges of the sick and the sterilization or destruction of things with which patients had been in contact. In his article "On the Contagion of Yellow Fever" he says:

Whenever a contagious disorder is attended with discharges that are characteristic of it, these discharges are always (as I have elsewhere endeavored to show) the chief vehicle of the morbid poison. They originate, in fact, in, and are the outward mark of, the very act of elimination. It is from this intimate connexion with the specific poison, in each particular case, that such discharges derive their specific character. I need scarcely add that yellow fever offers no exception to this law.

From the lofty heights of our present knowledge we may be tempted to smile at this positive statement, but we should bear in mind that up to 1901 similar views were held almost universally, and that at the time Budd wrote many still believed yellow fever to be non-communicable. His views were, of course, entirely academic, as he never saw the disease.

This cannot be said of his observations on tuberculosis. In his profession, which up to the time of his retirement was enormous, he had ample opportunity to study this disease both in its clinical and epidemiological aspects. The results of these studies appears in the form of a "Memorandum on the Nature and Mode of Propagation of Phthisis," which appeared in the *Lancet*, October 12, 1867 (page 451). This communication was made in an original and somewhat unusual way. This may best be explained by quoting the letter which G. E. Paget addressed to the *Lancet* on September 31, 1867, by way of an introduction to Budd's paper:

To the Editor of the Lancet:

SIR: The paper I send enclosed was received by me last December, in a sealed packet from Dr. W. Budd of Clifton, with a request that I would take charge of it until he should direct me to break the seal. At his desire I opened the packet a few days ago, and I now send you the contents, requesting the *favor* of their early publication in the *Lancet*. They are an epitome of what Dr. W. Budd has been for some time intending to publish in a more complete form; but his intention has been frustrated, and is still delayed, by the engrossments of professional practice and other circumstances beyond his control.

You will at once perceive the originality of his views and their very high importance, if established. If the evidence now given of their truth be incomplete, it is at least abundantly sufficient to raise them out of the mere hypothesis and ensure their careful consideration by pathologists.

In a letter to me Dr. W. Budd says, "I have strong reason for believing that my views on tubercle, with certain qualifications, apply to cancer also."

Budd's communication was simple and plain, but reflected in every line the intensity with which he had attacked the problem. He says:

The following are the principal conclusions to which I have been led regarding phthisis or tubercle:

1st. That tubercle is a true zymotic disease, of a specific nature, in the same sense as typhoid fever, scarlet fever, typhus, syphilis, etc., etc., are.

2nd. That like these diseases tubercle never originates spontaneously, but is perpetuated solely by the law of continuous succession.

3rd. That the tuberculous matter itself is (or includes) the specific morbific matter of the disease and constitutes the material by which phthisis is propagated from one person to another and disseminated through society.

4th. That the deposits of this matter are, therefore, of the nature of an eruption, and bear the same relation to the disease, phthisis, as the "yellow matter" of typhoid fever, for instance, bears to typhoid fever.

5th. That by the destruction of this matter on its issue from the body, by means of proper chemicals or otherwise, seconded by good sanitary conditions, there is reason to hope that we may eventually, and possibly at no very distant time, rid ourselves entirely of this scourge.

Time and space forbid the inclusion of the evidence on which he arrives at these conclusions. He says, however, that the idea of the communicability of tuberculosis occurred to him when he was taking a walk "in the second week of August, 1856." He remarks:

The long interval which has occurred between the summer of 1856 and the present time (1867) has been occupied in collecting data bearing on the question raised by this new theory—in accumulating evidence of various kinds and in examining and carefully weighing difficulties. I earnestly hope that they will not be too lightly rejected. At any rate, I can say that they have not been brought forward in haste or without due deliberation. I have, in fact, considerably exceeded the ten years which, with a fine sense of what is due to such an enterprise, the Roman poet prescribed as the time to be given to every composition intended by the writer to endure.

Laboratory studies and further epidemiological investigations have proven the correctness of Budd's conclusions—conclusions which are the more remarkable because they were made on clinical observations unsupported by experimental evidence and 15 years prior to the discovery of the tubercle bacillus.

His deductions on "Scarlet Fever and Its Prevention" (*British Medical Journal*, January 9, 1869, 1-23) might pass as the current views of to-day. He recognizes the value of disinfection and isolation, and decries the practice of frequent fumigation, which was then the accepted practice. He says:

There is good reason to believe that not only the eruption on the skin, but everything which is shed by the body of the infected, is heavily laden with the germs or seeds by which (alone, no doubt) the disease is propagated. The discharges from the throat and nose are, I imagine, especially virulent.

William Budd is described by his contemporaries as an eloquent orator—logical and gifted in debate. He was absolutely fearless in expressing the opinions which he held, yet ever modest and conservative. He clearly stated the results of his observations and made the deductions therefrom with logic and philosophy. In the British Medical Association, particularly in the Bristol branch, he occupied a leading place, and no aspect of medicine failed to attract the attention of his alert and energetic mind. His public health activities were unceasing. He acted as adviser to the Bristol health authorities, and on his own initiative investigated many epidemics of typhoid elsewhere and outlined the measures for their eradication. He issued, on several different occasions, pamphlets on the measures to be adopted for the prevention of the spread of typhoid fever and cholera, directions which would suit the needs of to-day very well. In his teaching and dispensary work he was ever busy; in fact, the accounts of his life show him to have been a man of such boundless capacity for work that his life was shortened by his indefatigable industry.

But to this incessant mental and bodily activity there had to be an end. He resigned the chair of the Practice of Medicine in the Bristol Medical School, and in 1862 gave up his appointment at the Bristol Royal Infirmary. Holidays in Germany and Switzerland would temporarily restore him to health, but his scientific energy was so unbounded that he would re-attack his work with such vigor that he would again break down. He suffered from agonizing headaches and his one desire was to complete his work on typhoid fever. This he did in 1873, and was immediately hurried away to France for rest and change. He returned to England in much worse health, and became (according to Clarke—British Medical Journal, 1880, I, 163-166) "partially hemiplegic." He died at Clevedon, on Friday, January 9, 1880, in his seventieth year. Early in life Budd married a Miss Hilton, a member of a well-known family in Kent, and had many children, by some of whom and his most devoted wife he was tenderly nursed until his death.

Budd's lasting contribution to the science of epidemiology may best be summed up in the summary of his book on typhoid fever. Other researches he made, to be sure, but many of these were contingent on his typhoid studies; studies which brought us nearer to the absolute prevention and eradication of this disease than the work of any other observer, either before or since his time. It, therefore, seems fitting that the words

which closed the *opus magnum* of his life, words which in reality closed the book of his scientific labors, should be the final chapter in this story of that pioneer epidemiologist, William Budd. The labors of a lifetime, and the ambitions of his career, are summed up in the following propositions:

1. That typhoid fever is, in its essence, a contagious or self-propagating fever, and is a member of the great natural family of contagious fevers, of which smallpox may be taken to be the type.

2. That the living body of the infected man is the soil in which the specific poison, which is the cause of the fever, breeds and multiplies.

3. That the reproduction of this poison in the infected body and the disturbance attaching to it constitute the fever.

4. That this reproduction is the same in kind as that of which we have, in smallpox, ocular demonstration.

5. That the disease of the intestine, which is its distinctive anatomical mark, is the specific eruption of the fever, and bears the same pathological relation to it which the smallpox eruption bears to smallpox.

6. That, as might have been anticipated from this view, the contagious matter by which the fever is propagated is cast off, chiefly, in the discharges from the diseased intestine.

7. That, as a necessary result, sewers and the cloacæ which, under existing sanitary arrangements, are the common receptacles of these discharges, are also the principal instruments in the transmission of the contagion; and, consequently, that in many instances the infected sewer, and not the infected man, appears as if it were the primary source of the specific poison.

8. That once cast off by the intestine, this poison may communicate the fever to other persons in two principal ways—either by contaminating the drinking water, or by infecting the air.

9. That, as an inevitable consequence of the impalpable minuteness of the contagious unit, and the many invisible and untraceable ways in which it is transmitted, cases must be constantly occurring, exactly as in the other contagious fevers, whose linear descent cannot be followed, and which spring up, therefore, under the semblance of spontaneous origin.

10. That the occurrence of such cases obviously constitutes no proof whatever that this fever ever does arise spontaneously.

11. That the exceeding speciality of the conditions attaching to the reproduction of the specific poison in the living body itself, as well as the facts relating to the geographical distribution, past and present, of this and the other contagious fevers, constitute evidence as strong as such evidence can ever be that none of these fevers originate spontaneously, but are propagated solely by the law of continuous succession.

And lastly—to crown the whole induction by a practical test—that by destroying the infective power of the intestinal discharges, by strong chemicals or otherwise, the spread of the fever may be entirely prevented, and that by repeating this process in every fresh case as it arises the disease may in time be finally extinguished.

In arriving at these conclusions, I would observe:

That the principal data on which they rest are, to the best of my belief, sure. Where these data consist in events observed by myself, I can vouch for their accuracy; where in events reported by others, I have taken, whenever this was possible, the most conscientious pains to verify them. In addition to this, all the great cardinal facts are distinguished in a peculiar degree by that precision and exactness which fit them alike for scientific statement and severe scientific deduction.

Whether the inferences drawn from the facts be logical, or whether, consistently with the laws of logic, any other inferences can be substituted for them, it is for others than myself to determine.

The whole induction is gradual. There are no great leaps, but the argument is conducted step by step; and I have striven to the utmost throughout to make one step sure before attempting the next.

The theory, in its entirety, is not only simple and harmonious, but it is in strict accordance with what we already know, of a certainty, of other members of the same family group.

Above all, its truth may be tested every day by a practical test, the employment of which can do no harm, and may do incalculable good.

Here I might fitly conclude. With this last step, as all may see, science passes into *duty*.

At this very hour scores of English homes are the scene of the agonizing anxieties with which recent experience has made the nation familiar. In hundreds, or, if we extend our view to the whole of Europe, in thousands, of cases, these anxieties have been consummated, within the month, by the death of the sufferer.

And all of this the work of an ignoble thing, which, if what has gone before be true, may be disarmed of its terrors by the simplest of precautions.

In my introductory remarks I endeavored to show, by illustrations drawn chiefly from the homes of the poor, how deeply humanity is concerned in the eradication of this fatal pest.

Since those remarks were first written (1859) the extension of its power to persons more conspicuous in rank has succeeded in awakening and directing, in a very striking way, the national attention to it. More than once since that time men or women bearing great historic names have fallen victims to it. Twice it has cast its dark shadow on the throne. Once it has placed the entire nation in mourning; and once again it has brought the entire nation, on bended knee, to the throne of grace in thanksgiving for a happier issue.

That the people of this kingdom should testify, in this most solemn of ways, their deep sense of responsibilities they have in this matter is well.

But if they wish to set a seal on the sincerity of their piety, there is—I say it with all humility—a still higher attitude to take.

It is, to spare from this hour no human effort to put under foot this great enemy of man. And let no one suppose that this is a matter in which he has no personal interest. The duty itself we may evade, but we can never be sure of evading the penalties of its neglect. This disease not seldom attacks the rich, but it thrives most among the poor. But by reason of our common humanity we are all, whether rich or poor, more nearly related here than we are apt to think. The members of the great human family are, in fact, bound together by a thousand secret ties, of whose existence the world in general little dreams.

And he that was never yet connected with his poorer neighbor, by deeds of charity or love, may one day find, when it is too late, that he is connected with him by a bond which may bring them both, at once, to a common grave.

DISCUSSION.

Dr. Hurd: I should like to call General Rucker's attention to the fact that Nathan Smith was a country practitioner in Vermont and New Hampshire between 1820 and 1830. He also studied typhoid fever, and said he had been familiar with cases for a number of years. He was strongly of the opinion that typhoid fever was contagious and was transmitted from one person to another. He had very clear ideas on the subject. I mention this to show another example of the opportunities afforded to the country practitioner to study disease.

Dr. Ford: Dr. Rucker's paper is one of great interest from a number of different standpoints and raises several points for consideration. In the first place, it seems to me that we should not attribute to such a county as Devonshire an ignorance which

is in any degree comparable to the ignorance which occurs in some of the backward parts of the United States. The county of Devonshire has an extremely interesting medical history. As far back as 1767, Dr. George Baker investigated the disease called Devonshire colic and palsy, and made probably the first definite contribution to our knowledge of industrial diseases, in that he was able to show that this colic and palsy in Devonshire was lead colic. Devonshire even at that time was noted for its milk and cider, the cider being highly prized in London. The people in Devonshire suffered greatly from colic and palsy, and it was not until George Baker went there and found that leaden vessels were employed in the manufacture of cider, and that Devonshire colic was lead colic, did the world have a correct idea of this condition.

It might be added that the Exeter Hospital is one of the oldest in England, having been established in the early part of the 18th century. Devonshire was also the seat of the first investigations upon the scientific disposal of sewage. The Cameron tank was discovered by a Scotchman who settled in Exeter. In modern times we have had a number of men from Devonshire who took a prominent part in the advancement of medical science in England.

In this country also we had a number of early observations upon cholera. Daniel Drake published his book on Epidemic Cholera in 1839. He enumerates all the causes which can be hypothecated for cholera, such as telluric influences, emanations from decaying matter and from infectious discharges. Although he saw several hundred cases of cholera during the years 1831-1833, he yet failed to grasp the essential thing in the constant association between new cases and old cases.

In Germany Pettenkofer, who occupied the stage especially from 1866 to 1886, which was the period shortly after Budd's final work in typhoid fever, was apparently not influenced in any way by Budd's observations. He always maintained that the material given off from a cholera or typhoid patient was not in itself infectious. That is the essential difference between the conclusions reached in Germany and by Budd and his followers in England. Pettenkofer maintained that this material must undergo a process of ripening in the soil before it is infectious and capable of communicating disease. Herein Budd stands as the great pioneer in the development of the science of epidemiology. It is of significance that when Koch developed bacteriology he at once came to the views concerning the epidemiology of disease which Budd had advocated.

It seems to me that it is especially interesting that whereas countries like Germany, Austria and France were for so many years unable to get rid of typhoid fever, England succeeded in doing it almost completely. For the last 20 years they have had only isolated cases. Sir Robert Boyce, who came over some years ago to investigate yellow fever, was surprised to learn how many cases of typhoid existed here. England, under the lead of men of the clearcut wisdom of Budd, has been able to eradicate typhoid fever, just as it has smallpox, by carrying out the strictest possible measures suggested by such individuals. In this country at the present time, and also in France and Germany, we are much behind England in this respect.

PROCEEDINGS OF SOCIETIES.

THE JOHNS HOPKINS HOSPITAL MEDICAL SOCIETY.

MARCH 6, 1916.

1. **Some Newer Methods in Rectal Surgery.** (Abstract.) DR. HARVEY B. STONE.

Dr. Stone presented informally a report of three procedures that are comparatively new and that he has been employing for the past several years.

(a) *Sacral Anæsthesia.*—This method was first introduced as a technique by Cathelin of Paris in 1902, but was first used for surgical anæsthesia by the Germans, notably Stoessel and Schlimpert. In this country it has been employed by Lynch of New York, and Bransford Lewis of St. Louis. The technique was described and a needle for the purpose of introducing a fluid was demonstrated. The needle is introduced into the sacral canal from below through the sacral hiatus. The fluid used is novocain in one per cent solution, which has been found more satisfactory than other substances experimented with. From 80 to 100 cc. have been employed. Good surgical anæsthesia is obtained by this method, but certain constitutional disturbances, as evidenced by rapid pulse rate and mental confusion, are met with occasionally. No ill after-effects were observed in the comparatively small number of cases in which this form of anæsthesia was employed by Dr. Stone and Dr. Hebb. In some cases the method is not feasible because of the difficulty in introducing the needle for anatomical reasons, and, in a small percentage of the cases in which the introduction is accomplished successfully, satisfactory anæsthesia fails to develop. The method thus offered, however, is a valuable aid to operation in the class of patients who would not be fit subjects for general anæsthesia, and who are suffering with conditions about the rectum and perineum not well adapted for the employment of local anæsthesia.

(b) *Report of a Method of Treating Pruritus Ani by the Injection of Alcohol.*—A paper will be published in the BULLETIN dealing with this subject in detail.

(c) *A New Operation for the Treatment of Recto-urethral Fistula.*—Dr. Young and Dr. Stone have operated upon seven cases of this exceedingly distressing and intractable condition with excellent results in every case. The older methods have been so unsatisfactory that the good results, so far obtained by the newer procedure, make its wider employment desirable. The method may be summarized as follows. First, the establishment of a suprapubic cystostomy to permit the drainage of urine away from the repair of the fistula. Second step, a midline incision in the perineum to the anterior margin of the anus, and a circular incision about the mucocutaneous margin of the anus. Through this incision the perineum is exposed, the sphincter ani muscle is divided in the midline anteriorly, and the rectal mucosa is dissected upward all around as in an exaggerated Whitehead operation. This dissection is carried upward so as to divide the fistulous tract, separating the rectal orifice from the urethra; and the rectum is then loosened from all its attachments above this point sufficiently far to permit the drawing down and out of the portion containing the rectal orifice of the fistula. The dissected bowel is then amputated through healthy tissue above the level of the fistula. The opening in the urethra is closed by sutures on a sound. Third step, repair. The perineal structures are brought together with sutures from side to side so as to separate the rectum from the urethra. The sphincter muscle is repaired. The stump of the bowel is sutured to the circular part of the skin incision, as in a Whitehead operation. The midline incision in the perineum is repaired with interrupted sutures.

2. **The Pathogenesis of Thyroid Intoxication.** DR. H. S. PLUMMER, Rochester, Minn.

DISCUSSION.

DR. L. F. BARKER: One cannot listen to anyone who has had such a large experience with exophthalmic goitre as Dr. Plummer has had without being deeply interested. I spent two days at

the Mayo Clinic in October last, most of the time with Dr. Plummer, and I was much interested in the kind of study going on there, especially in his careful statistical study of the cases.

It is necessary to understand the terms Dr. Plummer uses and to relate them to the terms in the bibliography, if we are to grasp the exact relation of the cases he describes to the cases that have been reported. You will notice Dr. Plummer speaks of hyperplastic goitre and non-hyperplastic goitre as two great subdivisions. By further subdividing, he describes the toxic and the atoxic forms of non-hyperplastic goitre. In the first place, this terminology is based upon pathological anatomy, but from his clinical experience he is able to correlate the clinical pictures with the pathological histology. In a very large number of cases, over 3000, he and his group of workers have been able to say beforehand from their clinical experience what cases belong to each group. That is indeed a remarkable clinical achievement.

Dr. Plummer uses the term "exophthalmic goitre" as practically synonymous with "hyperplastic goitre," as I understand him. Exophthalmic goitre is for him always a hyperplastic goitre. Non-hyperplastic toxic goitre is for him never "exophthalmic goitre." There is where his use of terms differs somewhat from the classifications used in the literature, inasmuch as for most writers "exophthalmic goitre" is used as a term synonymous with "Graves' syndrome," "Basedow's syndrome," and "hyperthyroidism." In other words, Dr. Plummer says that in "non-hyperplastic toxic goitre," there may be degrees of intoxication at times that yield symptoms which are almost indistinguishable from the symptoms that result from the intoxication in "hyperplastic goitre," or what he calls "true exophthalmic goitre." I think the difference in terminology is chiefly this—that Dr. Plummer reserves the term "exophthalmic goitre" for this one condition, namely hyperplastic goitre and its accompanying symptoms, a condition that runs a typical course clinically; whereas, the terms "Graves' disease," or "Basedow's disease," and for many the term "exophthalmic goitre," have been used in a wider sense to include not only "hyperplastic goitre," but also his "toxic non-hyperplastic goitre." If one keeps clearly in mind his use of terms, it is easy to follow Dr. Plummer's descriptions. Otherwise, one might be a little confused by trying to correlate his conceptions with those of contemporary medical literature.

One of the best things coming out of Dr. Plummer's work, it seems to me, is the very careful following of the course of these thyreopathies over long periods, by means of an analysis of records of careful observations. A thorough anamnesis is supported by "follow-up" records of the cases. Just as "jaundice" has, through the years, come to be subdivided into a series of conditions, so it would not be surprising if the *omnium gatherum* known as "Graves' disease," as "Basedow's syndrome," as "exophthalmic goitre," or as "hyperthyroidism" should be subdivided into a series of somewhat different conditions. This is what Dr. Plummer is trying to do, and he is giving names to the different members of the series and trying to differentiate them, so that we can all recognize them. I think this a very promising work and a kind for which we must all be grateful.

Much might be said about the theories advanced regarding the pathology of these conditions, the mode of action of the toxic substances upon the cells of the body, and the theories concerning iodine. These are all interesting topics and I should think that the ideas advanced may well serve as stimuli to a great deal of experimental work. As I understand it, they are to be regarded at present as simply working hypotheses, and we shall have to await results of experimentation before we can profitably discuss them at length.

Dr. Goetsch: I was very much interested in hearing what Dr. Plummer had to say in regard to the association between the histological picture and the clinical syndrome in thyroid disease. I do not know just what criteria Dr. Plummer has used with regard to the histological picture, but if they are the same as those which have been used during a good many years past, they are based largely upon the amount of colloid present in the thyroid, in the height of the epithelium and in the amount of hyperplasia generally of the alveolar cells. In general this association is undoubtedly true, for the large amount of colloid goes with the inactive type of thyroid. A small amount of colloid and a great amount of hyperplasia, a very cellular gland, go with marked hyperthyroidism.

There are a certain number of cases, however, in which this association does not hold. That is, there will be cases with a marked hyperthyroidism, in which, basing one's opinion upon these criteria, one would say the condition was not active. It is these cases that are difficult to explain upon the older histological basis. The index of the activity of the gland, therefore, will have to be based upon other than these criteria. In other words, one must know what the individual cells of the gland itself are doing. One can only determine this when the histological methods are so developed that there will be better criteria of activity than those previously accepted. Recently Dr. Bensley, of the University of Chicago, has developed a method by which he can demonstrate certain histological characteristics in the cells themselves which point to activity. The mitochondria are structures which are present in all cells and are present in numbers proportionate to the activity of the cell. For example, a cell which is undergoing active secretion or any cell which gives other evidences of activity, has a large number of these mitochondria present. If evidence of activity is associated with increased mitochondria content, then, when one is able to demonstrate increased mitochondrial content, one has a good basis for the assumption that these cells are active. When one applies these methods to certain cases of thyroid adenomata, we do find a very rich content in mitochondria. One has reason to feel that these cells with abundant mitochondria are active and are responsible for the symptoms present in these cases.

Then there is the adenoma with the capsule quite different from the surrounding thyroid tissue from which it can be shelled out without difficulty, the rest of the thyroid presenting every evidence of being normal. These cases show symptoms of typical hypertrophy, and when one examines a section of tissue taken from the thyroid surrounding one of these adenoma, and then the adenoma itself, one finds a striking difference in them, the normal thyroid presenting very few of the mitochondria and the adenoma cells being abundantly filled with them. It is well known that when one shells out one of these adenomata, symptoms subside rapidly.

In regard to the poisoning of the system. This is very interesting also. The thyroid toxæmia is undoubtedly the cause of the symptoms which the patients present. Just a word with regard to the effect upon the nervous system. Certain of these patients will show symptoms referable to activity of the sympathetic or autonomic nervous systems. It is interesting to see a patient come back after previous operation and to test her with the drugs which are calculated to stimulate one or the other of these systems. Recently I had occasion to examine a patient Dr. Halsted had operated on two years ago. She had forgotten altogether what her former symptoms were and she did not realize how much she had improved. When she received 10 mm. of adrenalin, she at once was transported back two years to her former condition. She said: "I feel now exactly like I felt each day two years ago. I realize now how much I have improved." This seemed to indicate that the thyroid secretion had an influence upon the syndrome which the adrenalin had in this instance. Similarly with the autonomic system, one can apply measures to bring out certain reactions which the patients show.

The hypertrophied gland does not merely secrete an abnormal amount of normal secretion. This is possibly the reason that the

symptoms are so varied. Moreover, it has been found that histologically there are structures present in the abnormal gland which are not present in the normal gland. If we can demonstrate histologically certain secretion antecedents in the hypertrophied cells that are not present in the normal ones, we have a certain basis for believing that we are dealing with a pathological secretion.

MARCH 20, 1916.

1. **Case of Auricular Flutter.** DR. C. H. GODDARD.

The case of auricular flutter to be shown to-night is of interest owing to its comparative rarity, and because of certain other exceptional features.

The patient, W. S., white, male, aged 61, came to the medical dispensary on March 14, 1916, complaining of shortness of breath. The history yielded the following points of interest.

At the age of 21 he had an attack of unconsciousness, which came on after he had run a considerable distance. It lasted for about two minutes, and left him none the worse for the experience. For 25 years there were no recurrences, but finally, 15 years ago, while working as a carpenter on a scaffold, he suddenly became faint and dizzy. Calling to a fellow workman, he collapsed, unconscious. This attack was again of about two minutes' duration, and was followed by no ill effects.

For three years he suffered no further inconvenience, but finally developed dyspnœa upon exertion, accompanied by frequent attacks of unconsciousness similar to those already described. Eight years ago the dyspnœa became so acute that he gave up his trade as carpenter, and became a night watchman. He nevertheless continued to have shortness of breath, and occasional attacks of unconsciousness.

One year ago he became a day watchman. This necessitated his being upon his feet most of the time, with the result that his dyspnœa became aggravated, and five months ago he gave up his work, since which time he has been living quietly at home. His last attack of unconsciousness previous to admission occurred five weeks ago, following the exertion of moving a heavy piece of furniture.

Physical examination was negative with the exception of a rather extensive pyorrhœa alveolaris, a moderate grade of emphysema, slight enlargement of the heart to the left, and a pulse rate of over 250. The urine was negative, and no signs of cardiac decompensation were apparent.

Further examination of the heart showed the P. M. I. to be in the fifth left interspace, 10½ cm. to the left of the median line. The relative cardiac dulness extended 11½ cm. to the left and 4 cm. to the right of the median line. The heart sounds were of a fœtal character, and the rate, as counted with a stethoscope at the apex, was about 250. As nearly as could be ascertained by palpation, the same rate prevailed at the wrist. Tracings were accordingly taken upon a Mackenzie ink polygraph.

Upon laying the patient down in preparation for this procedure, the pulse rate suddenly fell very markedly, and, when counted at the wrist and apex, was quite regular at 84 per minute. The veins of the neck, however, showed a rapid and rhythmical succession of large waves, which, when counted upon the tracing, were seen to be at the rate of 252 per minute.

In the tracing, the time-marker at the upper margin is beating five times to the second. Below this is the radial tracing at a rate of 84, and lowest of all the venous tracing upon which the auricular or "a" waves appear at a regular rhythm of 252 per minute, or exactly three times that of the radial pulse.

A number of tracings having been taken, the patient was dismissed, to return the following day for admission to the hospital. As he arose from the couch, the præcordium began to heave, and palpation of the pulse showed that the ventricular rate was again very high. A pulse tracing taken at once revealed a radial rate of 252. During the paroxysm the patient did not manifest any great discomfort, and dyspnœa was not marked. He described this and other attacks as "a feeling as though his heart were running away with itself."

He returned next day as directed, and was admitted to a medical ward. A tracing taken upon him within an hour or two after his admission showed an auricular rate of 264, with a ventricular of 132.

This was on March 15th. The 2 : 1 rate persisted until the following afternoon, as demonstrated by further tracings. At this time he was given 1 mg. of strophanthin intramuscularly, at 4 p. m. A tracing taken two hours later showed an apparent bradycardia, the arterial pulse registering but 60 beats to the minute. This condition varied with that of tachycardia, while an apex tracing cleared the matter up, showing apex impulses at a rate of about 120. At times these all arrived at the wrist with equal force; at others they barely showed upon the radial tracing, while intermediate phases existed between these two conditions. Therefore, we had at times a pronounced example of the "Pulsus Alternans."

As the therapeutic procedure in these cases is to give large doses of digitalis and strophanthus in an effort to produce auricular fibrillation, which may, in favorable cases, revert to the normal rhythm, a course of infusion of digitalis, in doses of two drachms four times daily for a few days, was begun on the evening of the 16th. On March 17th, the day following the strophanthin treatment, he continued to show an alternating pulse in greater or less degree, the apex rate remaining quite regular and fairly rapid.

On the following day, March 18, his auricles began to fibrillate. The irregularity in the radial rhythm is graphically demonstrated in the measurements of the time intervals between beats (in hundredths of a second) shown below the tracings. This condition persisted until noon of the following day, at which time one set of tracings, showed auricular fibrillation, while a second, taken immediately thereafter, revealed a normal, regular pulse with a prolonged conduction time between auricle and ventricle, the "a-c interval" measuring about .28″ instead of the usual .18″-.20″. Following this, the patient relapsed into fibrillation, and has persisted in this condition up to the present time.

In these cases, the symptoms, which include palpitation, dyspnœa, precordial distress, attacks of faintness, and sometimes even of unconsciousness, as in this case, are commonly followed by signs of cardiac decompensation. Some cases, however, go through attacks with little apparent discomfort and practically no signs of decompensation, as did this man. The danger lies in the fact that the continued rapid rate of the auricles, calling forth as it does very rapid ventricular contractions, may ultimately result in a profound embarrassment of the circulation, due to a weakening of the ventricular musculature under the unusual strain. The successful transformation of flutter into fibrillation, through active digitalis and strophanthus therapy, puts the heart in a condition where the ventricular rate may be readily controlled by the further administration of these drugs when necessary, and ordinarily results in a profound and rapid improvement in physical signs and symptoms. As the latter were not marked in this case, the onset of fibrillation of the auricles did not bring with it any marked change.

It is to be hoped that, therapy having been discontinued, the case will show itself to belong to that favorable group in which fibrillation is followed after a varying period of time by a normal rhythm, which persists. Other issues which commonly take place are: persistent fibrillation, relapse into flutter after a shorter or longer interval, and in some cases oscillations between auricular fibrillation, flutter, and normal rhythm. It is possible that oscillations between flutter and fibrillation existed during the early stages of fibrillation of the auricles in this case, as in some of the

tracings what appeared to be definite "a" waves appear upon curves where at other times no "a" waves are to be made out.

Care must be taken not to diagnose the onset of fibrillation in these cases from an irregular radial rhythm, without the support of other confirmatory evidence. This is due to the fact that the ventricles, instead of responding with regularity to every first, second, third or fourth auricular beat, as in this case, may respond successively now to the second, now the fourth, and now the third, and so on through many different combinations, giving a pulse that is irregularly irregular and hence very suggestive of fibrillation of the auricles.

It is to be noted that examination of the heart during the patient's stay in the hospital revealed no evidence of valvular defect. This makes for a favorable prognosis, which is admissible in such of these cases as present no evidence that the flutter is implanted upon lesions of valves or myocardium. The fact that the history indicates the probability that flutter has been present more or less constantly during the past 12 years, and the possibility that transient attacks of flutter occurred as long as 40 years ago, need also not militate against a satisfactory result. Cases are on record in which flutter has lasted for years, with ultimate recovery, while in others longer periods of years have elapsed, after the first appearance of symptoms, without serious injury to the patient.

2. **Further Studies on the Schick Reaction and Practical Active Immunization Against Diphtheria.** Dr. Wm. H. Park, New York City.

3. **Roentgenography as an Aid in the Localization of Cerebral Tumors. From a Study of 100 Cases.** Drs. G. J. Heuer and W. E. Dandy.

To appear later in the Bulletin.

THE JOHNS HOPKINS HOSPITAL HISTORICAL CLUB.

MARCH 13, 1916.

1. **The Superstition and Folklore of Menstruation.** Dr. Emil Novak.

To appear later in the Bulletin.

2. **William Budd, Pioneer Epidemiologist.** Dr. W. C. Rucker, U. S. Public Health Service.

This paper appears in full in the current number of the Bulletin.

THE LAENNEC.

MARCH 27, 1916.

1. **The Influence of Certain Organic Substances on the Growth of the Tubercle Bacillus.** Dr. P. A. Lewis, Philadelphia, Pa.

To appear later in the Bulletin.

2. **Experimental Meningitis.** Dr. C. R. Austrian.

To appear later in the Bulletin.

NOTES ON NEW BOOKS.

A Clinical Study of the Serous and Purulent Diseases of the Labyrinth. By Dr. Erich Ruttin. With a foreword by Professor Dr. Victor Urbantschitsch. Authorized translation by Horace Newhart, A. B., M. D. Cloth, $2.00 net. (*New York: The Rebman Company, 1914.*)

Dr. Ruttin was one of Politzer's assistants and is still connected with the University Clinic in Vienna. For many years this clinic has been particularly interested in the labyrinth and Dr. Ruttin's contributions, together with those of his associates Borony, Neumann, Beck, and Urbantschitsch, have entirely revised the old opinions concerning the physiology and pathology of the inner ear.

This volume has been very intelligently translated by Dr. Newhart and is invaluable for those interested in the functional tests of the hearing and vestibular apparatus, and in the pathology and theraphy of the inner ear.

S. J. C.

A Manual of Diseases of the Nose and Throat. By Cornelius G. Coakley, A. M., M. D. Fifth edition. (*Philadelphia: Lea & Febiger, 1914.*)

This fifth edition of Dr. Coakley's manual, although in some respects an improvement on former editions, still leaves much to be desired from the point of view of the fourth year medical student.

Take for example the chapter on diseases of the tonsil. From it the student can get a very good idea of the clinical picture of a pharyngitis, acute tonsilitis and diphtheria, but only a very poor idea of the diagnosis of the chronic insidious infections of the tonsils, which are of so much importance in the etiology and treatment of many of the general systemic disorders—cervical adenitis, arthritis, digestive disturbances, renal lesions, anæmia, etc.

Furthermore, the advice concerning the operative procedures is not in accord with the principles of general surgery. This is particularly true in the directions given for controlling hemorrhage after the removal of the tonsils.

For the specialist, on the other hand, the book contains many valuable suggestions concerning the diagnosis and treatment of the common maladies of the throat and larynx.

S. J. C.

The Detection of Poisons and Powerful Drugs. By Dr. Wilhelm Autenreith. Authorized translation by William H. Warren, Ph. D. Cloth, $2.00. (*Philadelphia: P. Blakiston's Son & Co., 1914.*)

This is the translation by William H. Warren, Professor of Chemistry in Wheaton College, of the standard laboratory manual on the detection of poisons, written by Wilhelm Autenrieth and now in its fourth edition. The best methods for the recognition of the various poisons are given in considerable detail and quantitative methods are elaborated for certain substances like arsenic, the presence of which in excess is evidence of crime. The book is well written, carefully translated, and will prove of great value to medical men interested particularly in legal medicine.

W. W. F.

Infant Feeding—Its Principles and Practice. By F. L. Wachenheim, M. D. Cloth $2.00. (*Philadelphia: Lea & Febiger, 1915.*)

In recent years many books have appeared dealing with the feeding of infants and written for a professional audience. They aim to do two things, first, to give the methods employed for feeding infants in health and disease; and second, to point out the reasons why certain methods are advantageous and certain others disadvantageous, and to set forth the principles which should govern the choice of any one particular food or the variation of the different food elements. The first aim is usually well accomplished, the second very rarely so.

In this book the author discusses the various problems met with in feeding infants and gives methods and various formulæ. The book reflects well the views of current authors, particularly those in Germany. These views are faithfully followed, even to

the employment of the unsatisfactory classification of Finkelstein for diseases of the gastrointestinal tract. The subject matter is presented in a concise but comprehensive manner, and the summary enables one to readily determine the facts essential for the successful feeding of infants. The book can be recommended as a practical handbook for infant feeding; it does not deal extensively with the principles of the nutrition of infants, but outlines the practice.

Lectures on the Heart. By THOS. LEWIS, M. D., F. R. C. P., D. Sc. $2.00 net. (*New York: Paul B. Hoeber, 1915.*)

In the autumn of 1914 Dr. Lewis visited America. The five lectures he delivered during his stay make up the small book entitled "Lectures on the Heart." In the first, the Harvey lecture in New York, he gives an excellent portrayal of the basic principles of electrocardiography, with a summary of the work that his laboratory has developed in the demonstration of the origin and course of "The Excitation Wave in the Heart." The next three lectures are those of the Herter Foundation, presented before the members of The Johns Hopkins University. The subjects are "The Method of Electrocardiography Exemplified"—an elementary discussion of the information acquired by the application of electrocardiography to the study of cardiac irregularities; "The Relation of Auricular Systole to Heart Sounds and Murmurs"—an interesting presentation of cardiophonography with a detailed discussion of auricular sounds and the different types of murmurs present in mitral stenosis; "Observations upon Dyspnœa with Especial Reference to Acidosis"—a résumé, perhaps somewhat theoretical, of his latest experiments in the chemical laboratory on the diminished alkalinity of the blood with excess of non-volatile acids and acid salts, as an additional cause for dyspnœa. The fifth lecture, on "Observations upon Cardiac Syncope," was given at the opening of the Faculty of Medicine at McGill. In this he groups the different causes of cardiac syncope and gives proof of the actual physiological and anatomical basis for such grouping. Besides their medical instruction, these lectures all emphasize a basis maxim held by Dr. Lewis—"the application of laboratory methods to questions of immediate and practical consequence."

It is rare that one has the opportunity to read so delightful a presentation, as Dr. Lewis here offers, of clear-cut scientific deductions. The combination of experimental research with accurate clinical observation, withal expressed so clearly and forcibly, is almost unique. Everywhere between the lines are numerous suggestions for those interested in this line of work. After reading the first lecture on the "Excitation Wave in the Heart," one despairs of finding a more stimulating experimental discussion with such sharply defined results. For this chapter alone every medical man should have this book.

E. W. B.

Infection, Immunity and Specific Therapy. By JOHN A. KOLMER, M. D., PH. D. pp. 870. Cloth, $6.00. (*Philadelphia: W. B. Saunders Co., 1915.*)

This work is the most valuable one of its kind in English literature. The author has clearly stated the purposes of the volume: "First, to give practitioners and students of medicine a connected and concise account of our present knowledge regarding the manner in which the body may become infected, and the method in turn by which the organism serves to protect itself against infection or strives to overcome the infection, if it should occur, and also to present a practical application of this knowledge to the diagnosis, prevention, and treatment of disease; second, to give to physicians engaged in laboratory work and special workers in this field a book to serve as a guide to the various immunologic methods; third, to outline a laboratory course in experimental infection and immunity for students of medicine and those especially interested in these branches."

It might seem at first sight that the accomplishment of these three purposes within one book would be a matter well-nigh impossible; yet even a casual review must convince one, not only of the author's complete grasp of the subjects under consideration, but also of his admirable clearness in the presentation of the various subjects he takes up. The book is divided into five main parts. Part 1 deals with general immunologic technique and includes descriptions of how to make suitable pipettes, the washing of blood corpuscles, methods for obtaining human and animal blood, the technique of animal inoculation and the preservation of the sera, etc.

Under Part 2 is included the general subject of infection and the principles governing it, so far as we know them.

Part 3 takes up the principles of immunity and immunological technique from the practical standpoint. Thus, in this section one finds admirable chapters on the opsonic index, the preparation of bacterial vaccines, agglutinins, antitoxins, complement-fixation reactions, anaphylaxis, etc.

Five chapters in Part 4 treat of the application of the principles of immunity in the prophylaxis, diagnosis and treatment of disease.

The last section includes a series of 60 different exercises designed to constitute a thorough course in experimental infection and immunity.

The book is essentially practical. Though the theoretical side of the various topics is well handled, greater emphasis has been laid upon the presentation of the application of the theory in practice. It would be very hard to pick out any one section which is better than another, for the reason that all the various methods are given a place in the book, the author's personal experience with reference to them is concisely stated, but in unbiased terms, and the proof of the superiority of this or the other method is left entirely for the reader or worker to prove for himself. A splendid feature of the book is shown in the frequent italicized sentences, which make it possible for the casual reader to pick out the points which the author is particularly anxious to emphasize. It should be pointed out that the author has in every case described the various tests and reactions in great detail, thus tending to secure accuracy, simplicity and definiteness. No particular attempt has been made to embody in the book all the literary references, but from time to time the more important ones are cited. Numerous well-colored illustrations, frequent tables, sample protocols of titrations, etc., play a large share in making the book a useful daily companion for the laboratory worker. There is also included an account of those immunologic, diagnostic reactions which have a direct bearing upon veterinary medicine. It is difficult to see how the book could be improved upon. It can, therefore, be recommended to students, physicians and laboratory workers as the one giving the best presentation of the subjects dealt with at present in existence in English literature.

S. R. M.

JOHNS HOPKINS HOSPITAL BULLETIN.

The Hospital Bulletin contains details of hospital and dispensary practice; abstracts of papers read and other proceedings of the Medical Society of the Hospital, reports of lectures, and other matters of general interest in connection with the work of the Hospital. It is issued monthly. Volume XXVII is now in progress. The subscription price is $2.00 per year. (Foreign postage, 50 cents.) Price of cloth-bound volumes, $2.50 each.

A complete index to Vols. I-XVI of the Bulletin has been issued. Price 50 cents, bound in cloth.

BOOKS RECEIVED.

International Clinics. A Quarterly of Illustrated Clinical Lectures and Especially Prepared Original Articles. By leading members of the medical profession throughout the world. Edited by Henry W. Cattell, A. M., M. D. Volume IV. Twenty-fifth series. 1915. 8°. 384 pages. J. B. Lippincott Company, Philadelphia and London.

Progressive Medicine. A Quarterly Digest of Advances, Discoveries and Improvements in the Medical and Surgical Sciences. Edited by Hobart Amory Hare, M. D. Assisted by Leighton F. Appleman, M. D. Volume IV. December, 1915. 8°. 421 pages. Lea & Febiger, Philadephia and New York.

The Adolescent Period: Its Features and Management. By Louis Starr, M. D., LL. D. 1915. 12°. 211 pages. P. Blakiston's Son & Co., Philadelphia.

The Physiology of the Amino Acids. By Frank P. Underhill, Ph. D. 1915. 12°. 169 pages. Yale University Press, New Haven; Humphrey Milford, Oxford University Press, London.

The Clinics of John B. Murphy, M. D., at Mercy Hospital, Chicago. Volume IV, Number 6. December, 1915. W. B. Saunders Company, Philadelphia and London.

A Text-Book of Physiology. By William H. Howell, Ph. D., M. D., Sc. D., LL. D. Sixth edition, thoroughly revised. 1915. 8°. 1043 pages. W. B. Saunders Company, Philadelphia and London.

Practical Cystoscopy and the Diagnosis of Surgical Diseases of the Kidneys and Urinary Bladder. By Paul M. Pilcher, A. M., M. D. Second edition, thoroughly revised, with 299 illustrations, 29 in colors. 1915. 8°. 504 pages. W. B. Saunders Company, Philadelphia and London.

Bone-Graft Surgery. By Fred. H. Albee, A. B., M. D., F. A. C. S. With 332 illustrations, three of them in colors. 1915. 8°. 417 pages. W. B. Saunders Company, Philadelphia and London.

Post-Mortem Examinations. By William S. Wadsworth, M. D. With 304 original illustrations. 1915. 8°. 598 pages. W. B. Saunders Company, Philadelphia and London.

Question Manual. Compiled by May Kennedy, R. N. 1915. 8°. 158 pages. Whitcomb & Barrows, Boston.

Oxford Medical Publications. Publishers: Henry Frowde, London; Hodder & Stoughton, London. The following 4 volumes:

1. *A Guide to Gynæcology in General Practice.* By Comyns Berkeley, M. A., M. D., M. C. (Cantab.), F. R. C. P. (Lond.), and Victor Bonney, M. S., M. D., B. Sc. (Lond.), F. R. C. S. (Eng.), M. R. C. P. (Lond.). 1915. 8°. 452 pages.
2. *Medical Lectures and Aphorisms.* By Samuel Gee, M. D. With recollections by J. Wickham Legg. 1915. 12°. 408 pages.
3. *Diseases of the Throat, Nose and Ear.* By William H. Kelson, M. D., B. S., F. R. C. S. (Eng.). 1915. 8°. 270 pages.
4. *Instinct and Intelligence.* By N. C. Macnamara, F. R. C. S. 1915. 12°. 216 pages.

Injuries of the Eyes, Nose, Throat and Ears. By Andrew Maitland Ramsay, M. D., F. R. F. P. S. (Glasgow), J. Dundas Grant, M. D., F. R. C. S. (Eng.), H. Lawson Whale, M. D. (Camb.), F. R. C. S. (Eng.), and Charles Ernest West, F. R. C. S. (Eng.). 1915. 16°. 160 pages. Oxford War Primers.

Report on the Medico-Military Aspects of the European War. From Observations Taken Behind the Allied Armies in France. By Surgeon A. M. Fauntleroy, U. S. Navy. Under the direction of the Bureau of Medicine and Surgery, Navy Department, Washington, D. C. 1915. 8°. 146 pages. Government Printing Office, Washington.

Interstate Commerce Commission. Twenty-Ninth Annual Report. Part I. 1915. 8°. 188 pages. Government Printing Office, Washington.

Saint Thomas's Hospital Reports. New series. Volume XLII. 1913. Edited by J. J. Perkins and C. A. Ballance. 1915. 8°. 205 pages. J. & A. Churchill, London.

THE INSTITUTIONAL CARE OF THE INSANE IN THE UNITED STATES AND CANADA.

BY

HENRY M. HURD, WILLIAM F. DREWRY,
RICHARD DEWEY, CHARLES W. PILGRIM,
G. ALDER BLUMER, T. J. W. BURGESS.

Edited by HENRY M. HURD, M. D.

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